











46 I

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## ORIGINAL CONTRIBUTIONS.

|  | PAGE |
|--|------|
| Progressive Ankylotic Rigidity of the Spine, by B. Sachs and J. Fraenkel.....  | 1    |
| Meralgia Paresthetica (Roth), with the Report of Ten Cases, by John Herr Musser and Joseph Sailer.....   | 16   |
| The Unity of the Acute Psychoses, by Philip Coombs Knapp.....  | 41   |
| Two Cases of Muscular Dystrophy with Necropsy, by Wm. G. Spiller.....  | 47   |
| A Study of the Lesions in a Second Case of Trauma of the Cervical Region of the Spinal Cord, Simulating Syringomyelia, by James Hendrie Lloyd.....   | 65   |
| Landry's Paralysis, by Philip Coombs Knapp and John Jenks Thomas.....  | 74   |
| A Case of Hematomyelia, by James Hendrie Lloyd.....  | 92   |
| A Case of Tumor at the Base of the Brain in the Pontine Region, by James Hendrie Lloyd.....  | 103  |
| Anniversary Address, by Wharton Sinkler.....   | 104  |
| The Nervous Equivalent of Fever, by Henry S. Upson.....  | 104  |
| The Relation Between Trigeminal Neuralgias and Migraine, by Jas. J. Putnam.....  | 129  |
| Lesion of the Optic Chiasm with a Clinical Report of Three Cases, by Wm. M. Leszyansky.....  | 135  |
| Epileptic Ambulatory Automatism, by D. J. McCarthy.....  | 143  |
| Landry's Paralysis. Remarks on Classification, by W. E. Taylor and J. E. Clark.....  | 177  |
| A Case of Unilateral Progressive Ascending Paralysis, Probably Representing a New Form of Degenerative Disease, by Chas. K. Mills.....   | 195  |
| A Case Presenting Right Sided Hemiplegia with Hemianesthesia, Right Homonymus Hemianopsia, Jargon Aphasia, Wernicke's Pupillary Reaction Sign and Neuritic Pain in the Arm of the Paralyzed side, by F. X. Dercum..... | 201  |
| A Case of Acute Poliomyelitis Anterior in a Youth Seventeen Years of Age, by Wharton Sinkler.....  | 205  |
| Transient Real Blindness in Hysteria, by Geo. C. Harlan.....   | 209  |
| Inaugural Address Before the American Neurological Association, by Edward D. Fisher.....   | 241  |
| Tumor of the Superior Parietal Convolution Accurately Localized and Removed by Operation, by Chas. K. Mills and W. W. Keen. With a Pathological Report on the Nature of the Growth, by Wm. G. Spiller.....             | 244  |
| A Case of Amaurotic Family Idiocy, by Hugh T. Patrick.....   | 265  |
| A Case of Amaurotic Family Idiocy, by Sydney Kuh.....  | 268  |
| The Appearance of the Fundus Oculi, by Chas. H. Beard.....   | 270  |
| Raynaud's Disease in the Insane, by J. E. Courtnev.....  | 272  |
| Two Cases of Muscular Atrophy of the Peroneal Type, by Given Campbell.....   | 274  |
| Contribution to the Study of the Plantar Reflex, Based upon Seven Hundred Examinations Made With Special Reference to the Babinski Phenomenon, by G. L. Walton and W. E. Paul.....                                     | 305  |
| Two Cases of Primary Neurotic Atrophy, Bearing Resemblance to Multiple Neuritis, by F. X. Dercum.....  | 324  |

|  | PAGE |
|--|------|
| A Digest of Recent Work on Epilepsy, by L. Pierce Clark.....   | 331  |
| A Case of Wernicke's Conduction Aphasia with Autopsy, by<br>Howell T. Pershing.....  | 369  |
| A Clinical Study of Some Reflexes, by Joseph Fraenkel and Jo-<br>seph Collins.....   | 375  |
| A Digest of Recent Work on Epilepsy, by L. Pierce Clark.....   | 387  |
| Autopsy in a Case of Adiposis Dolorosa with Microscopical Ex-<br>amination, by F. X. Dercum.....   | 419  |
| Imperative Ideas in the Sane and their Management, by Edward<br>B. Angell.....   | 430  |
| Christian Pseudo-Science and Psychiatry, by Smith Baker.....   | 438  |
| A Case of Monocular Hysterical Amaurosis in a Girl Eleven Years<br>of Age, by C. A. Veasey.....  | 444  |
| On the Use of a New and Efficient Hypnotic in the Treatment of<br>the Insane: Chloretone, by J. Percy Wade.....  | 447  |
| The Anatomic-Cytological Relationship of the Neurone to Disease<br>of the Nervous System, by L. F. Barker.....   | 469  |
| The Pathological Changes in the Neurone in Nervous Disease, by<br>Wm. G. Spiller.....  | 487  |
| How Does the Neurone Doctrine Affect the Conception of Ner-<br>vous Disease? by B. Sachs.....  | 506  |
| A Case of Adiposis Dolorosa with Necropsy, by Chas. W. Burr.....   | 519  |
| The Functional Significance of the Size and Shape of the Neu-<br>rone, by Henry H. Donaldson.....  | 526  |
| Revised Interpretation of the Central Fissures of the Educated<br>Suicide's Brain, by Burt G. Wilder.....  | 537  |
| Rigidity of the Spine, by James H. McBride.....  | 542  |
| A Case of Primary Progressive Muscular Dystrophy of the Facio-<br>Scapulo-Humeral Type of Landouzy and Dejerine, by Allen<br>B. Bonar.....                         | 547  |
| A Case of Hysterical Aphonia in a Grand Mal Epileptic, by<br>L. Pierce Clark.....  | 551  |
| Summary of the Results Obtained in One Hundred and Four-<br>teen Miscellaneous Cases of Nervous Disease, by F. X.<br>Dercum.....                                   | 569  |
| Two Cases of Tumor of the Spinal Cord, by John Jenks Thomas.....   | 580  |
| Reconstruction of Serial Sections of Brains, by Adolf Meyer.....   | 600  |
| Experimental Work on Brain Pressure Following Injury, by Wm.<br>N. Bullard.....  | 601  |
| A Form of Subacute Pressure—Neuritis, by Theo. H. Kellogg.....   | 603  |
| A Case of Multiple Neuritis with Atrophy; Fibrillary Twitchings,<br>Cramps and Exaggerated Reflexes: Two Years' Duration<br>and Recovery, by Wm. James Morton..... | 605  |
| A Case of So-called Landry's Paralysis with Autopsy, by Sidney<br>I. Schwab.....   | 619  |
| An Atypical Case of Multiple Sclerosis, by Chas. W. Burr and<br>D. J. McCarthy.....  | 634  |
| A Case of Malaria Presenting the Symptom of Disseminated<br>Sclerosis with Necropsy, by Wm. G. Spiller.....  | 643  |
| Notes Upon the Occurrence of Multiple Neuritis and Beri-Beri in<br>Alabama, by E. D. Bondurant.....  | 645  |
| Section of the Posterior Spinal Roots for the Relief of Pain in a<br>Case of Neuritis of the Brachial Plexus, by Morton Prince.....                                | 649  |

## INDEX OF AUTHORS' NAMES.

Figures in parentheses ( ) indicate discussions. Figures with asterisk \* indicate original articles, and are accompanied with title. Figures unaccentuated, accompanied with title, indicate abstracts; without title, book reviews.

| PAGE  | PAGE   |
|---|--|
| Adamkiewicz, A.—Ganglion Cells .....300   | Binswanger, O.....235  |
| Adler, I.—Artificial Fixation of Joints in Tabes.....600  | Bischoff, E.—Motor Tracts—Origin of Epileptic Attacks.. 57   |
| Angear, J. M.....295  | Bodoni, P.—Bromopine in Epilepsy.....365   |
| Angell, E. B.—Imperative Ideas in the Sane.....*430, 442, 532   | Bodey, G. W.—Sinus, Case of, June, p. xxiv.  |
| Anton, G.—Cortical Blindness and Deafness .....651  | Boettiger, A.—Acromegaly and Raynaud's Disease.. .....463  |
| Auerbach, S.—Acute Anterior Poliomyelitis.....227   | Bolton, P. ....(214)   |
| Babes, V.—Use of Brain Substance in Nervous Disease..615  | Bonar, A. B.—Progressive Muscular Dystrophy .....*547  |
| Bailey, P.—(151); Diplegia Brachialis Traumatica....214, (281)  | Bondurant, E. D.—Multiple Neuritis and Beri-Beri.....*645  |
| Baker, W. A.—Nervous Prostration, May, p. xxx.  | Brady, E. F.—Epilepsy, June, p. xxiv.  |
| Baker, S.—Christian Pseudo-Science and Psychiatry 438, (442)  | Brester—Paramyoclonus and Epilepsy.....561   |
| Barachini, V.—Traumatic Tetanus.....516   | Brissaud, E.—Variable Chorea.122   |
| Barbacci, O.—Anatomical, Physiological and Pathological Relationship of Nerve Cell..219   | Brower, D. R.—Neurological Observations in Hawaiian Islands .....170   |
| Barker, L. F.—174; Anatomic-Cytological Relationship of the Neurone to Nervous Disease .....469, (534)  | Brown, C. H.—Therapeutical Experiences in Diabetes Mellitus, Feb., p. xxii.  |
| Barker—Unusual Gunshot Wound of Brain .....413  | Bullard, W. N.—Experimental Work on Brain Pressure Following Injury .....*601  |
| Bayerthal—Brain Tumor.....515   | Burr, C. B.....(294), (554)  |
| Beard, C. H.—Appearances of the Fundus Oculi.....270  | Burr, C. W.—(220); Acute Internal Hydrocephalus, 223, (224), (349); Adiposis Dolorosa,*519; Treadler's Palsy, 556; General Anesthesia, 557, (558). |
| Bechterew, v. W.—Scapulo-Humeral Reflex, 404; Objective Symptoms of Local Hyperesthesia and Anesthesia, 412; Myotonia a Disease of Metabolism.....612 | Burr, C. W., and McCarthy, D. J.—Multiple Sclerosis .....*634  |
| Benoit, F.—Abdominal Tympanites of Hysterical Origin .....358   | Campbell, G.—Muscular Atrophy. ....*274  |
| Berger, H.—Anatomy of Cerebral Cortex .....302  | Caro—Relations Subsisting Between Epileptic Attacks and Excretion of Uric Acid.....661   |
| Berkley, H. J.—(349); Sensory Anesthesia Without Psychological Implications .....351  | Caswell, W. H.—Progressive Muscular Dystrophy, (653), 656, (659)   |
| Betteremieux, P.—Neuralgias and Tics of Face—Relation with Pathologic Condition of Lachrymal Ducts .....229   | Cestan, R.—Syphilitic Polyneuritis .....468  |
|   | Chambreleut—Epilepsy and Pregnancy .....517  |

- |   | PAGE        |   | PAGE       |
|---|-------------|---|------------|
| Church, A.—Symptoms of Brain Tumor—Differential Diagnosis .....   | 457         | Donadieu-Lavit — T a b e s and Traumatism .....   | 225        |
| Claiborne, J. H., Jr.—Amaurotic Family Idiocy.....  | 664         | Donaldson, H. H.—Functional Significance of Size and Shape of the Neurone.....  | 526, (540) |
| Clark, J. E.—Landry's Paralysis .....   | *177        | Donath, J.—Traumatic Periodic Paralysis.....  | 517        |
| Clark, L. P.—Digest of Recent Work on Epilepsy, *387; Hysterical Aphonia in Grand Mal Epileptic .....   | *551        | Du Four, H.—Tubercular Polyneuritis .....   | 446        |
| Clarke, J. M.—Loss of Knee-jerk in Paralyzed Limbs in Hemiplegia .....  | 226         | de Buck, D.—Erb's Syndrome.....   | 408        |
| Cohn, M.—Toe-Reflex .....   | 102         | de Montyel, M.—Malarial Infection and Epilepsy.....   | 361        |
| Collins, J.—(43), (51); Rigid Spine, 52, (54), (106), (110); Syringomyelia, 150; Tumor of Aqueduct of Sylvius, 151, (154), (157); Myelitis, 218, (282), (284), (285); Clinical Study of Reflexes....  | *375, (533) | Edinger, L.—Connection of Sensory Cranial Nerves with Cerebellum—Direct Sensory Cerebellar Tract. ....  | (59)       |
| Conner—Multiple Sclerosis....   | (654)       | Elliott, G. R.....  | (53)       |
| Corwin, A. M.....   | 126         | Ellis, H.—Evolution of Modesty .....  | 446        |
| Courtney, J. E.—Raynaud's Disease .....   | *272        | Eshner, A. A.—(22); Case of Myxedema, 123, (220), (287), (290); Rhizomelic Spondylosis .....  | 291        |
| Couvelaire, A., and Marie, P.—Generalized Neurofibrosis.....  | 415         | Etienne, G.—Progressive Muscular Atrophy—Myelopathic Origin .....   | 299        |
| Crocq, J.—Differential Diagnosis—Hysterical Apoplexy —Organic Apoplexy .....  | 55          | Eulenburg, A.....   | 368, (556) |
| Cunningham, R. H., (151), (156), (655)  |             | Farnarier, F.—Acromegaly and Mental Degeneracy .....  | 456        |
| Curtis, B. F.—Thyroidectomy for Graves' Disease, 405; Resection of Sympathetic for Graves' Disease .....  | 405         | Feinburg, J. A.—Asthenic Bulbar Paralysis .....   | 613        |
| D'Alloco—Myoclonus .....  | 562         | Féré, A.—Tolerance of Elderly Epileptics to Bromides, 102; Accidents of Epileptic Paroxysm with Muscular Contraction .....  | 612        |
| Dana, C. L.—(52), (152); Hemorrhagic Encephalitis and Myelitis.....   | 152, (216)  | Féré, Ch.—Pruritus and Trichotillomania in General Paresis .....  | 55         |
| Dercum, F. X.—(42), (115), Acute Poliomyelitis, 116, (121); Myopathy, 158; Syphilitic Paraplegia, 162; Clinical Case, *201; Neurotic Atrophy, 287, (289), (292); Cerebral Diplegia, 293; Primary Neurotic Atrophy, *324, (356), (385); Autopsy in Case of Adiposis Dolorosa, *419, (453); Paramyotonus, 454, (532), (554), (555), (556), (557); Rhizomelic Spondylosis, 558; Studies in Astereognosis ..... | *560        | Fickler, A.—Compression of Cord in Vertebral Caries.....  | 231        |
| Dewey, R.—Drug Addiction....  | 294         | Finkelnburg—Paralysis of Peroneal Nerve in Tabes.....   | 230        |
| Dold, W. E.....   | (295)       | Fisher, E. D.—(101), (154), (156); Amyotrophic Lateral Sclerosis, 213, (214); Amer. Neurol. Assoc. Address, *241, (406), (408), (653), (654), (657), (659)  |            |
|   |             | Flatau, E.....  | 61         |
|   |             | Flatau, E., and Jacobsohn, L.....   | 304        |
|   |             | Fraenkel, J.—Ankylotic Rigidity of the Spine, *1; Disease of Thoracic Division of Sympathetic Chain, 282; Clinical Study of Reflexes, *375, (408); Congenital Hydrocephalus, 653, (656), (657), (660) |            |



|   | PAGE       |   | PAGE              |
|---|------------|---|-------------------|
| Francine, A. P.—Tabes Dorsalis in Negroes.....  | 349        | Hirsch, Wm.—Multiple Neuritis and Acromegaly, 50, (51), (52); Dysphrenia, 110; Paralysis of Serratus Magnus—Glosis of Mid-Dorsal Segments.. | 215               |
| Freund, C. S., and Sachs, H.  | 467        | Hoche—Pathology of Spinal Cord After Embolus.....   | 414               |
| Frolich—Severe Skull and Brain Injury .....   | 411        | Hofmann, A. — Meningo-Typhus..  | 667               |
| Friedmann, M.—Spastic and Syphilitic Spinal Paralysis....   | 233        | House, W.—Etiology and Pathology of Major Epilepsy....  | 662               |
| Fuchs, A.—Observation of Sensory Jacksonian Epilepsy....  | 610        | Hyslop, J. H.—Newspaper Science..   | 208               |
| Gasne, G.—Hysterical Anorexia .....   | 412        | Jaboulay—Treatment of Trophic Lesions of Foot.....  | 194               |
| Gasne, G., and Gullain, G.—Segmentary Angioma .....   | 466        | Jacobi, M. P.—(111), (214); Exophthalmic Goitre..   | 652               |
| Gerhardt, D.—Differential Diagnosis—Mutiple Sclerosis ..  | 228        | Jacobsohn, L....  | 61                |
| Gibert—Tabetic Arthropathies and Radiography .....  | 564        | Janet, P., and Raymond, F.—Tic of Foot.....   | 518               |
| Gibney, V. P.....(51), (52)   |            | Janeway, T....  | (405)             |
| Goethe, J. H.—Nervous Prostration, July, p. xxii.   |            | Jeliffe, S. E.....  | 304. (367)        |
| Gordinier, H. C.....  | 367        | Jones, R.—Paralysis of the Serratus Magnus..  | 40                |
| Gonzales and Verga—Epilepsy Associated with Paramyoclonus .....   | 552        | Josserand, M.—Pseudo-Hypertrophic Paralysis..   | 664               |
| Gould, A. P., and Warren, J. C.....   | 416        | Kalischer, S.—Telangiectasia with Unilateral Hypertrophy.   | 297               |
| Gowers—Minor Epilepsy .....   | 361        | Kazowsky, A. D.—Acute Delirium..  | 232               |
| Gradle, H.—Neurosis .....   | 409        | Keen, W. W.—Tumor of Superior Parietal Convolution..  | *244              |
| Grassmann, K. — Intermittent Claudication .....   | 664        | Kellogg, T. H.—Subacute Pressure—Neuritis..   | *603              |
| Gray, L. L.—Enuresis Nocturna, June, p. xxiv.   |            | Keraval, P., and Raviart, G.—Huntingdon's Chorea..  | 667               |
| Greeno, H. S.—Treatment of Chorea, May, p. xxx.   |            | Kiernan, J. G.....  | (171)             |
| Grohmann, A.—Experiences in Employment for Nervous People .....   | 364        | Kirkpatrick, E. A.—Development of Voluntary Movement..  | 386               |
| Guillian, G.—Double Amyotrophic Paralysis .....   | 429        | Klein, S.—Anæmia Pseudoleukæmia, Jan., p. xxx.  |                   |
| Guillain, G., and Gasne, G.—Segmentary Angioma .....  | 466        | Knapp, P. C.—Unity of Acute Psychoses, 41, (45); Landry's Paralysis..   | *74. (438), (442) |
| Hammond, G. M.—(83); Multiple Sclerosis—Progressive Muscular Atrophy, 106, (151), (156); Intracranial Tumor, 216, (281), (285); Word Deafness.. | 406, (406) | Koppen, M.—Acute Paranoia..   | 546               |
| Hammill, S. McC.—Migraine with Aphasia .....  | 117        | Kraepelin—Psychical Effects of Alcohol..  | 330               |
| Harlan, G. C.—Transient Real Blindness in Hysteria.....   | *209       | Kreкке—Scoliosis Ischiadica..   | 358               |
| Hauser, G.—Cerebral Commotion..   | 667        | Kuh, S.—Amaurotic Family Idiocy, *268, (295); Cerebral Localization..   | 460               |
| Heidenhain, A.—Acquired Internal Acute Hydrocephalus.   | 122        | Ladame, P., and von Monakow, C.—Aneurism of Left Vertebral Artery..   | 565               |
| Hinshelwood, J.....   | 303        | Langdon, F. W..(90), (539), (644)   |                   |

- |  | PAGE                                      |  | PAGE  |
|--|---|--|---|
| Lange—Etiology of Epilepsy..   | 610                                       | Marinesco—Changes in Large<br>Pyramidal Cells..  | 73  |
| Lannois, M.—Post-Paroxysmal<br>Albuminuria in Convulsive<br>Epilepsy, 91; Retropulsive<br>Epilepsy..   | 204                                       | Meier, G. H.—Acute Rheuma-<br>tism, Mar., p. xxx.  |   |
| Lannois and Carrier—Analge-<br>sia of Ulnar Nerve in Epi-<br>leptics..   | 518                                       | Meige, H.—Chronic Hereditary<br>Tropho-Edema..   | 296   |
| Laymen, H. M.....  | (171)                                     | Meirowitz, P.—Intracranial<br>Growth, 107; Hysterical Dis-<br>turbances of Vision.....   | 285   |
| Lamboth—Trephining for Trau-<br>matic Epilepsy..   | 566                                       | Mellus, E. L.....  | (169)   |
| Lamprasi—Hemi-craniectomy<br>for Epilepsy..  | 566                                       | Meyer, A.—Reconstruction of<br>Serial Sections of Brains,<br>(540), 600, (650)   |   |
| Lenznick—Spinal Cord Injur-<br>ies..   | 614                                       | Meyer, E.—Sarcoma of Third<br>Ventricle, Metastases of<br>Fourth.....  | 565   |
| Lermoyez, M.—Hysterical Nas-<br>al Insufficiency..   | 419                                       | Mills, C. K.—(41), (119), (168);<br>Unilateral Progressive As-<br>cending Paralysis, *195, (221),<br>Tumor of Superior Parietal<br>Convolution.....  | *244, (290),<br>(541), (557), (559), (600), (643) |
| Letchworth, W. P.....  | 617                                       | Mingazzini, G.—Pupillary Re-<br>flexes..   | 123   |
| Leszynsky, W. M.—(51); Les-<br>ions of the Optic Chiasm,<br>*135; Facial and Retrobulbar<br>Neuritis, 150, (156), (216);<br>Meirowitz—Paresthetic Me-<br>ralgia.....   | 216, (219), (281),<br>(284), (285), (406) | Mirallie—Post-Traumatic Con-<br>vulsions—Essential Epilepsy<br>—Craniectomy..  | 611   |
| Leutert—Cerebral Abscess—<br>Operation..   | 464                                       | Mitchell, J. K.....  | (556)   |
| Linde, M.—Hemianopsia..  | 414                                       | Mitchell, S. W.....  | (439)   |
| Lindstom, A. H.—Treatment<br>of Pain in Tabes Dorsalis...  | 366                                       | Monro, T. K.....   | 417   |
| Lloyd, J. H.—Trauma of Cer-<br>vical Region of Spinal Cord,<br>Simulating Syringomyelia,<br>*65; A Case of Hematomye-<br>lia, *92; Tumor at Base of<br>Brain in Pontine Region<br>*103, (119), (221), (291), (557) |   | Moore, W. E.....   | 303   |
| Lowenfeld—Neurotrophic Cu-<br>taneous Disturbances .....   | 46  | Moremen, J. S.—Derangement<br>of Liver and Disease, Jan.,<br>p. xxiv.  |   |
| Luxenburg, J.—Morphological<br>Changes in Cells of Anterior<br>Horn of Cord.....   | 668                                       | Morgan, F. P.—Cerebral Rheu-<br>matism..   | 361   |
| Luce, H.—Nothnagel's Convul-<br>sive Center in Pons of Man..   | 56  | Morton, W. J.—Multiple Neu-<br>ritis with Atrophy-Fibrillary<br>Twitchings—Cramps—Exag-<br>gerated Reflexes..  | *605  |
| Lyman, H. M.....   | 95  | Moyer, H. N.....   | (172)   |
| Mailhouse, M.—Hereditary Cer-<br>ebellar Ataxia..  | 658                                       | Muhlig—Epileptiform Attack<br>During Convalescence from<br>Typhoid Fever..   | 611   |
| Maltschauoff—Gonococcus Tox-<br>ine—Action on Nervous Sys-<br>tem..  | 230                                       | Musser, J. H.—Meralgia Pa-<br>resthetica..   | *17   |
| Manclaire, P.—Cerebral and<br>Spinal Commotions. ....  | 173                                       | Munsterberg, H.—Psychologi-<br>cal Atomism..   | 211   |
| Margulies, A.—Failure of<br>Knee-Jerk in Lesions of Up-<br>per Spinal Cord.....  | 296                                       | McBride, J. H.—Rigidity of<br>Spine..  | *542  |
| Marie, P., and Couvelaire, A.—<br>Generalized Neurofibrosis....  | 415                                       | McCarthy, D. J.—Ambulatory<br>Automatism, 115, (121); Epi-<br>leptic Ambulatory Automat-<br>ism, *143, (169); Acute In-<br>ternal Hydrocephalus, 223,<br>(287); Trophic Changes in<br>Distribution of First Branch<br>of Fifth Nerve Following |   |

|   | PAGE       |  | PAGE                            |
|---|------------|--|---------------------------------|
| Traumatism, 288; Paresthetic Meralgia, 289, (290); Narcolepsy..   | 363, (453) | Putnam, J. J.—Relation Between Trigeminal Neuralgias and Migraine..*   | 129, (428), (441), (527), (553) |
| McCarthy, D. J., and Burr, C. W.—Multiple Sclerosis ..  | *634       | Putnam, J. W.....  | (44)                            |
| McCaffrey—Treatment of Acute Peritonitis, Sept., p. xxiv.   |            | Raecke—General Paralysis and Tabes..   | 112                             |
| Nancrede, C. B.....   | 127        | Raimann, E.—Question of Retrograde Degeneration.....   | 614                             |
| Newboldt, G. P.—Paralysis of the Serratus Magnus.....   | 40         | Raviart, G., and Keraval, P.—Huntingdon's Chorea .....   | 667                             |
| Nouges, E., and Sirol, J.—Mental Torticollis..  | 514        | Raymond, F., and Janet, P.—Tic of Foot.....  | 518                             |
| Noyes, W. B.—Aneurism of Aortic Arch.....   | 281        | Redlich, E.—126; Pseudologia Phantastica, 463; Senile Epilepsy..   | 560                             |
| Obersteiner, H.....   | 126        | Remak, E.—Femoral Reflex—Dorsal Region—Spinal Cord..   | 297                             |
| Oberwarth—Syphilitic Pseudo-paralysis.....  | 157        | Ribot—Epileptic Myoclonus.....   | 525                             |
| Onuf, B.—(102), (108), (154); Lead Paralysis with Autopsy, 155; Ophthalmoplegic Migraine.....   | 215        | Riggs, C. E.....   | (91)                            |
| Packard—Acromegaly..  | 453        | Romme—Treatment of Epilepsy.....   | 566                             |
| Patrick, H. T.—(42), (101); Brain Tumor, 172; Amaurotic Family Idiocy, *265; Hereditary Cerebellar Ataxia.....  | 409        | Rothmann, M.—Cataleptic Lethargy.....  | 357                             |
| Paul, W. E.—Planter Reflex, *305; Epilepsy.....   | 359        | Roux, J. C.....  | 567                             |
| Pearce, F. S.—(115), (120); Artisan's Palsy, 149; Melancholy and Hypochondriasis, 160; Epileptic Ambulatory Automatism, 161, (222), (224), (288), (290), (349), (455); Peripheral Facial Palsy—Crossed Hemiparesis..... | 553        | Russell, J. W.—Inquiry Into Prevalence of Epilepsy—Relation to Other Disease.....  | 359                             |
| Penrose, C. B.....  | 418        | Sachs, B.—Ankylotic Rigidity of the Spine, *1, (51), (52), (106); Tumor Compressing the Cauda Equina, 108, (111); Transplantation of Tendons—Infantile Cerebral Palsy, 121, (217); Specific Cervical Pachymeningitis, 281, (283), (440); How Does the Neurone Doctrine Affect the Conception of Nervous Disease..... | 506, (535)                      |
| Peters—Dormiol: A New Hypnotic.....   | 608        | Sachs, H., and Freund, C. S.....   | 467                             |
| Peterson, F.—Amaurotic Family Idiocy....51, (54), (110), (112), (406), (654), (655), (656)  |            | Sailer, J.—Meralgia Parasthetica .....   | *17, (356)                      |
| Pershing, H. T.—Wernicke's Conduction Aphasia.....  | *369       | Sano, F.—Progressive Amyotrophy.....   | 271                             |
| Pick, F.—Progressive Muscular Dystrophy.....  | 663        | Sante de Sanctis.....  | 60                              |
| Piltz, J.—Pupillary Reflexes...200  |            | Savill, T. D.....  | 124                             |
| Piollet—Congenital Tumor...374  |            | Schlapp, M. G.....(214), (219)   |                                 |
| Porter, W. H.—Raynauds Disease.....(653), 660   |            | Schlesinger—Relation of Acromegaly to Diabetes Mellitus..  | 464                             |
| Potts, C. S.—Muscular Dystrophy, 113; Injury of Spinal Cord, 113, (357), (368); Amyotrophic Lateral Sclerosis...556   |            | Schroder—Diffuse Sarcomatosis.....   | 169                             |
| Prince, M.—Neuritis of Brachial Plexus.....   | 649        | Schultze — Epileptic Equivalents.....  | 609                             |
| Pritchard, W. B.....(660)   |            | Schultze, F.—Surgical Treatment—Tumors of Spinal Membranes .....   | 226                             |
| Pryor, W. R.....  | 304        | Schwab, S. I.—Landry's Paralysis with Autopsy.....   | *619                            |

|   | PAGE         |  | PAGE         |
|---|--------------|--|--------------|
| Shallcross, W. G.—Acromegaly.....   | 554          | Thomas, H. M.....  | (49)         |
| Sicard, A.—Subarachnoidal Injections.....   | 365          | Thomson, St. C.....  | 125          |
| Sinkler, W.—Anniversary Address, 105, (116); Acute Poliomyelitis Anterior, (121), *205, (222), (288); Parkinson's Disease.....  | 350          | Thompson, H. B.—Total Number of Functional Cells in Cerebral Cortex of Man.....  | 563          |
| Sirol, J., and Nougès, E.—Mental Torticollis.....   | 514          | Thorndike, E. L.....   | 60           |
| Sklarek, F.—Congenital Myxedema.....  | 15           | Townsend, W. R.....  | (53)         |
| Soca, F.—Prolonged Sleep—Caused by Tumor of Hypophysis.....   | 666          | Trenel—Amnesic Aphasia.....  | 560          |
| Sollier, P.—Paresthetic Neuralgia.....  | 286          | Troemmer, E.—Internal Ophthalmoplegia in Ophthalmoplegic Migraine.....   | 230          |
| Souques, A.—Meralgia Paresthetica, 56; Double Syndrome of Weber—Autopsy.....  | 466          | Upson, H.—Nervous Equivalent of Fever.....   | (42), 105    |
| Spiller, Wm. G.—(44); Muscular Dystrophy with Necropsy, 47, (101), (115); Relation of Migraine to Epilepsy, 118, (165); Amyotrophic Lateral Sclerosis, 156; Tabetic Symptoms and Exaggerated Knee-Jerks, 158, 220, (223); Pathological Report on Tumor in Parietal Convolution, *245, (287), (290); Atypical Tremor—290; Unilateral Internal Hydrocephalus, 293, (350), (356); Brain from Case of Congenital Absence of Visual Tracts, 357, (453), (454); Landry's Paralysis, 455; Pathological Changes in the Neuron in Nervous Disease, 487, (534), (540), (554); Internal Hydrocephalus, 559; Malaria—Case of..... | 643, (643)   | Veasey, C. A.—Monocular Hysterical Amaurosis.....  | *444         |
| Spratling, W. P.—Epilepsy.....  | 661          | Verga and Gonzales—Epilepsy Associated with Paramyoclonus.....   | 552          |
| Stefani, A.—Irritability.....   | 348          | Vigouroux, A.—Dystrophic Edema.....  | 243          |
| Stelwagon, H. W.....  | 61           | Vogt—Occupation Treatment in Functional Neurosis.....  | 364          |
| Stewart, J. A.—Enuresis Diurna et Nocturna, June, p. xxiv.  |              | Van Gehuchten, H.....  | 174          |
| Stieglitz, L.....   | (151), (156) | Van Gehuchten, A.—General Symptoms of Paraplegia—Compression of Spinal Cord, 228; Phenomena of Repair in Central Nervous System..... | 233          |
| Still, G. H.—Occurrence of Day Terrors in Children.....   | 298          | Van Gieson.....  | (528)        |
| Stintzig, R.—Relation of Heart Disease to Epilepsy.....   | 642          | v. Hosslin—Syphilis of Brain.....  | 665          |
| Taylor, E. W.—Landry's Paralysis.....   | *177         | van Walsen, G. C.—Weight of Heaviest Brain Yet Described.....  | 644          |
| Terriberly.....   | (51), (52)   | von Monakow, C., and Ladame, P.—Aneurism of Left Vertebral Artery.....   | 565          |
| Thomas, J. J.—Landry's Paralysis, *74; Tumor of Spinal Cord.....  | *580         | Wade, J. P.—Chloretone.....  | *447         |
|   |              | Walton, G. L.—Plantar Reflex.....  | *305, (386)  |
|   |              | Warren, J. C., and Gould, A. P.....  | 416          |
|   |              | Weightman, W. A.—Amenorrhea—Dysmenorrhea, July, p. xxiv.   |              |
|   |              | Wiener, R. G.....  | (215), (285) |
|   |              | Wilder, B. G.—Revised Interpretation of Central Fissures of Educated Suicide's Brain.....  | 537          |
|   |              | Wilder, W. H.—Value of Eye, Ear Symptoms in Brain Tumor.....   | 460          |
|   |              | Wilkin, W. B.—Treatment of Torticollis.....  | 284          |
|   |              | Wille—Epidemic Neuralgia.....  | 225          |
|   |              | Wilson, T. S.—Chorea.....  | 234          |
|   |              | Witmer, A. F.—Hemiplegia with Partial Claw Hand.....   | 287          |
|   |              | Ziehen, T.....   | 127          |

# INDEX OF SUBJECTS.

Figures in parentheses ( ) indicate discussions. Figures with asterisk \* indicate original articles, and are accompanied with title. Figures unaccented, accompanied with title, indicate abstracts.

| PAGE   | PAGE  |
|--|---|
| Abdominal Tympanites of Hysterical Origin .....358                         | Anthropological—Bibliography, 30; Feb., p. xxiv.              |
| Acromegaly—Atypical Case of, 453, 554                                      | Aortic Arch Aneurism.....281                                  |
| Acromegaly and Mental Degeneracy .....456                                  | Aphasia—Amnesic .....560                                      |
| Acromegaly and Raynaud's Disease .....463                                  | Aphasia with Migraine.....117                                 |
| Acromegaly and Diabetes Mellitus .....464                                  | Aphasia—Jargon .....*201                                      |
| Acromegaly—Bibliography, 37; June, p. xx.                                  | Aphasia—Wernicke's Conduction—Autopsy. ....*369               |
| Adiposis Dolorosa—Autopsy in Case of.....*419, (428)                       | Aphasia—Bibliography, 39; July, p. xx.                        |
| Adiposis Dolorosa with Necropsy .....*519                                  | Aphonia in Epileptic.....*551                                 |
| Alcohol—Psychical Effects of.....330                                       | Apoplexy—Hysterical and Organic—Differential Diagnosis. 55    |
| Amaurotic Family Idiocy, 51, *268, 664; Bibliography, 38; June, p. xxvi.   | Apoplexy—Temperature in.....226                               |
| Amaurosis—Monocular Hysterical.....*444                                    | Astereognosis—Studies in.....*569                             |
| American Neurological Association—Inaugural Address.*241                   | Asthenic Bulbar Paralysis.....613                             |
| Amnesic Aphasia .....560   | Ataxia—Bibliography, 41; Sept., p. xviii.                     |
| Amyotrophic Lateral Sclerosis, 165, 213, 556                               | Ataxia—Hereditary Cerebellar, 409, 658                        |
| Amyotrophic Paralysis—Double 429   | Ataxia—Locomotor .....*576                                    |
| Amyotrophy—Progressive — of Traumatic Origin .....271                      | Ataxic Paraplegia. ....*577                                   |
| Analgesia of Ulnar Nerve in Epileptics .....518                            | Atomism—Psychological .....211                                |
| Anatomic—Cytological Relationship of Neurone to Nervous Disease .....*469  | Atrophy—Muscular.....106, *274                                |
| Anatomical Physiological and Pathological Relations of Nerve Cell. ....219 | Atrophy—Multiple Neuritis with *605                           |
| Anatomy of Cerebral Cortex—Finer.....300                                   | Atrophy—Multiple Sclerosis with Progressive Muscular .....106 |
| Anatomy—Bibliography, 29; Jan., p. xx.                                     | Atrophy—Progressive Muscular —of Myelopathic Origin.....299   |
| Anesthesia—General .....(557)  | Atrophy—Primary Neurotic.. 287, *324                          |
| Anesthesia and Hyperesthesia —Objective Symptoms of...412                  | Auto-Toxics and Toxics in Production of Epilepsy.....*338     |
| Anesthesia—Pathological Findings in Case of Cutaneous Sensory .....351     | Automatism—Epileptic Ambulatory .....*143, 161                |
| Aneurism of Aortic Arch.....281  | Beri-Beri. ....134  |
| Aneurism of Left Vertebral Artery .....565                                 | Beri-Beri and Multiple Neuritis.....*645                      |
| Angioma—Segmentary. ....466  | Blindness—Cortical—and Deafness .....651                      |
| Anniversary Address .....104   | Books Received.....64, 240, 468                               |
| Anorexia—Hysterical .....412   | Brain—Bibliography, 31; Mar., p. xx.                          |
|  | Brain, Congenital Absence of Visual Tracts .....357           |
|  | Brain—Educated Suicide's *537, (539)                          |
|  | Brain Injury—Severe Skull and 411                             |
|  | Brain Pressure—Experimental Work on .....601                  |
|  | Brain—Reconstruction of Serial Sections of.....600            |
|  | Brain Substance—Use of—In Nervous Disease .....615            |

|                                 | PAGE        |                                     | PAGE       |
|---------------------------------|-------------|-------------------------------------|------------|
| Brain—Syphilis of.....          | 665         | Chronic Hereditary Tropho—          |            |
| Brain—Unusual Gunshot           |             | Edema.....                          | 296        |
| Wound of.....                   | 413         | Chyluria—Simulation of.....         | 357        |
| Brain—Weight of Heaviest yet    |             | Classification—Landry's Paraly-     |            |
| Described.....                  | 644         | sis.....                            | *177       |
| Brain Tumor—Case of...*         | 103, 172    | Claudication—Intermittent.....      | 664        |
| Brain Tumor—Differential Di-    |             | Convulsions—Essential Epi-          |            |
| agnosis.....                    | 457         | lepsy.....                          | 611        |
| Brain Tumor—General Symp-       |             | Cord Injury—Spinal.....             | 113        |
| toms of.....                    | 457         | Cord—Trauma of.....                 | 614        |
| Brain Tumor—Value of Eye and    |             | Cortex—Cells in.....                | 563        |
| Ear Symptoms in.....            | 460         | Cortex-Cerebral—Finer Anat-         |            |
| Bibliography, 36; May, p. xxvi. |             | omy of.....                         | 302        |
| Bromides—Tolerance of Elder-    |             | Cranial Nerves—Bibliography,        |            |
| ly Epileptics to.....           | 602         | 29; Jan., p. xx; Bibliography,      |            |
| Bromipine in Epilepsy.....      | 365         | 47, Dec., p. xviii.                 |            |
| Bulbar Paralysis—Asthenic.....  | 613         | Cutaneous Sensory Anesthesia        |            |
| Cataleptic Lethargy.....        | 357         | —Pathological Findings in           |            |
| Cauda Equina, Tumor.....        | 108         | Case of.....                        | 351        |
| Cells in Cortex.....            | 563         | Cytology—Bibliography, 47;          |            |
| Cells—Changes in—of Anterior    |             | Dec., p. xviii.                     |            |
| Horn of Cord.....               | 668         | Day-Terrors in Children—Oc-         |            |
| Cells—Changes in Large Pyra-    |             | currence of.....                    | 298        |
| midal.....                      | 73          | Degeneracy—Acromegaly and           |            |
| Cells—Ganglion.....             | 300         | Mental.....                         | 456        |
| Cells—Pyramidal Giant—Ob-       |             | Degeneration—Retrograde.....        | 614        |
| servations on Changes of.....   | 486         | Delirium—Pathological Anat-         |            |
| Central Fissures—Interpreta-    |             | omy and Bacteriology of             |            |
| tion of.....                    | *537, (539) | Acute.....                          | 232        |
| Cerebellum—Tumor of.....        | 151         | Diplegia.....                       | *576       |
| Cerebellar Tumor—Bibliog-       |             | Diplegia Brachialis Traumatica..... | 214        |
| raphy, 45; Nov., p. xviii.      |             | Diplegia—Cerebral—Case of.....      | 293        |
| Cerebellar Ataxia—Hereditary,   |             | Diabetes Mellitus and Acrome-       |            |
| 409, 658                        |             | galy.....                           | 464        |
| Cerebral Abscess with Opera-    |             | Dormiol—A New Hypnotic.....         | 608        |
| tion.....                       | 464         | Drug Addiction.....                 | 294        |
| Cerebral Commotion.....         | 667         | Dysphrenia.....                     | 110        |
| Cerebral Cortex—Finer Anat-     |             | Dystrophic Edema.....               | 243        |
| omy of.....                     | 300         | Dystrophy—Muscular.....             | 47, 113    |
| Cerebral Diplegia—Case of.....  | 293         | Dystrophy—Primary Progress-         |            |
| Cerebral Cortex—Total Num-      |             | ive Muscular.....                   | *547       |
| ber of Functional Cells in—     |             | Dystrophy—Progressive Mus-          |            |
| of Man.....                     | 563         | cular.....                          | 656        |
| Cerebral Localization.....      | 460         | Edema—Dystrophic.....               | 243        |
| Cerebral Rheumatism.....        | 361         | Embolus—Pathology of Spinal         |            |
| Cerebral and Spinal Commo-      |             | Cord After.....                     | 414        |
| tions.....                      | 173         | Encephalitis and Myelitis—          |            |
| Cervical Pachymeningitis—Spe-   |             | Hemorrhagic.....                    | 152        |
| cific.....                      | 281         | Epidemic Neuralgia.....             | 225        |
| Chiasm—Lesions of Optic...*     | 135         | Epilepsy.....                       | 359, 661   |
| Chicago Neurological Society,   |             | Bibliography, 29; Jan., p. xx;      |            |
| 170, 294, 457                   |             | Bibliography, 45; Nov., p. xvii.    |            |
| Chloretone.....                 | *447        | Epilepsy—Bromipine in.....          | 365        |
| Chorea—Bibliography, 31; Mar.,  |             | Epilepsy—Diagnosis and Men-         |            |
| p. xx.                          |             | tal States in.....                  | *389       |
| Chorea—Huntingdon's.....        | 667         | Epilepsy—Digest of Recent           |            |
| Chorea—Treatment of—by Sul-     |             | Work on.....                        | *381, *387 |
| phocarbolate of Soda.....       | 234         | Epilepsy—Etiology of...*            | 331, 610   |
| Chorea—Variable.....            | 122         |                                     |            |

|  | PAGE      |   | PAGE     |
|--|-----------|---|----------|
| Epilepsy—Etiology and Pathology of.....                  | *394, 662 | Facial Palsy—Peripheral .....   | 553      |
| Epilepsy — Hemi-Craniectomy for.....                     | 566       | Facial and Retrobulbar Neuritis.....  | 150      |
| Epileptic—Hysterical Aphonia in .....                    | *551      | Fundus Oculi—Appearances of .....   | *270     |
| Epilepsy—Jacksonian.....                                 | 610       | Fracture Vertebra and Cord.....   | 614      |
| Epilepsy and Malarial Infection.....                     | 361       | Ganglion Cells .....  | 300      |
| Epilepsy—Manifestations of.....                          | *342      | General Anesthesia.....   | (557)    |
| Epilepsy—Major—Etiology and Pathology of.....            | 662       | General Paralysis and Tabes.....  | 112      |
| Epilepsy—Minor .....                                     | 361       | General Paresis .....   | 55       |
| Epilepsy and Paramyoclonus, .....                        | 552, 561  | Bibliography, 35; May, p. xx.   |          |
| Epilepsy—Post-Paroxysmal Albuminuria in Convulsive.....  | 91        | Generalized Neurofibrosis .....   | 415      |
| Epilepsy—Post-Traumatic.....                             | 611       | Goitre—Exophthalmic .....   | 652      |
| Epilepsy and Pregnancy.....                              | 517       | Gonococcus Toxine—Action on Nervous System .....                              | 230      |
| Epilepsy—Prognosis in.....                               | *390      | Graves' Disease—Resection of Sympathetic for.....                             | 405      |
| Epilepsy—Senile.....                                     | 560       | Graves' Disease—Thyroidectomy for .....                                       | 405      |
| Epilepsy—Surgical Treatment of.....                      | *394      | Hammond, Dr. William A.....   | 157      |
| Epilepsy—Relation of Heart Disease to.....               | 642       | Hemorrhagic Encephalitis and Myelitis .....                                   | 152      |
| Epilepsy—Relation to Other Diseases .....                | 359       | Hematomyelia—A Case of.....   | *93      |
| Epilepsy—Relation of Migraine to.....                    | 118       | Hemianopsia in One Eye.....   | 414      |
| Epilepsy—Retropulsive.....                               | 204       | Hemianopsia—Right Homonymous .....  | *201     |
| Epilepsy—Toxics and Auto-Toxics in Production of.....    | *338      | Hemianesthesia — Right-Sided Hemiplegia with .....                            | *201     |
| Epilepsy—Treatment of.....                               | *391, 566 | Hemi-Craniectomy for Epilepsy .....   | 566      |
| Epilepsy—Treatment of—with Bromides.....                 | 615       | Hemiplegia with Hemianesthesia—Right-Sided.....                               | *201     |
| Epilepsy—Trephining for Traumatic.....                   | 566       | Hemiplegia with Partial Claw-Hand.....  | 287      |
| Epileptic Ambulatory Automatism.....                     | *143, 161 | Hereditary Cerebellar Ataxia.....   | 409      |
| Epileptics—Analgesia of Ulnar Nerve in.....              | 518       | Hereditary Tropho-Edema — Chronic.....  | 296      |
| Epileptic Attacks.....                                   | 661       | Homonymous Hemianopsia—Right.....   | *201     |
| Epileptic Attacks—Origin of.....                         | 57        | Hyperesthesia and Anesthesia —Objective Symptoms of.....                      | 412      |
| Epileptic Equivalents.....                               | 609       | Hypertrophy — Telangiectasia with Unilateral .....                            | 297      |
| Epileptic Paroxysm—Accidents of .....                    | 612       | Hypnotic—Dormiol.....   | 608      |
| Epileptic Predisposition .....                           | *334      | Hypochondriasis — Melancholy .....  | 160      |
| Epileptic Myoclonus .....                                | 525       | Hydrocephalus—Acute .....   | 122      |
| Epileptic—Tolerance of Elderly —to Bromides.....         | 602       | Hydrocephalus—Acute Internal .....  | 223, 559 |
| Erb's Syndrome.....                                      | 408       | Hydrocephalus—Congenital .....  | 653      |
| Eruetation.....  | 443       | Hydrocephalus—Unilateral Internal.....  | 293      |
| Etiology of Acute Anterior Poliomyelitis.....            | 227       | Hysteria—Transient Real Blindness in.....                                     | *209     |
| Etiology of Epilepsy.....                                | *331, 610 | Hysteria and Tympanites.....  | 358      |
| Etiology—Paresthetic Meralgia with Trauma .....          | 289       | Hysteria — Bibliography, 30; Feb., p. xxvi; Bibliography, 43; Oct., p. xviii. |          |
| Exophthalmic Goitre, 652; Bibliography, 40; Aug., p. xx. |           | Hysterical Amaurosis—Monocular.....   | *444     |

PAGE

PAGE

Hysterical Anorexia. . . . . 412  
 Hysterical Aphonia in Epilep-  
 tic. . . . . \*551  
 Hysterical—Disturbances of Vis-  
 ion—Curious. . . . . 285  
 Hysterical Nasal Insufficiency. 410  
 Hysterical and Organic Apo-  
 plexy—Differential Diagnosis 55  
 Idiocy—Amaurotic Family, 51,  
 \*268, 664  
 Idiocy—Bibliography, 44; Oct.,  
 p. xx.  
 Imperative Ideas in Sane. . . . \*430  
 Insane—Raynaud's Disease in  
 the. . . . . \*272  
 Internal Hydrocephalus—Uni-  
 lateral. . . . . 293  
 International Medical Congress 63  
 Intracranial Tumor. . . . . 107, 216  
 Injections—Subarachnoidal . . 365  
 Injury—Severe Skull and Brain. 411  
 Irritability. . . . . 348  
 Jacksonian Epilepsy . . . . . 610  
 Jargon Aphasia. . . . . \*201  
 Knee-Jerks—Case with Tabetic  
 Symptoms and Exaggerated. 159  
 Knee-Jerk in Lesions of Upper  
 Cord—Researches on Failure  
 of . . . . . 296  
 Landry's Paralysis. . . . . \*74, (90), 454  
 Landry's Paralysis with Au-  
 topsy. . . . . \*619  
 Landry's Paralysis—Remarks on  
 Classification. . . . . \*177  
 Lateral Columns—Systemic Dis-  
 ease of. . . . . 541  
 Lead Paralysis. . . . . 155  
 Lethargy—Cataleptic . . . . . 357  
 Localization—Cerebral. . . . . 460  
 Locomotor Ataxia. See Tabes.  
 Locomotor Ataxia. . . . . \*576  
 Malaria—Case of. . . . . 643, (643)  
 Malarial Infection and Epilepsy 361  
 Manifestations of Epilepsy. . \*342  
 Melancholia—Bibliography, 33;  
 April, p. xx.  
 Melancholy and Hypochondri-  
 asis. . . . . 160  
 Meningo—Typhus. . . . . 667  
 Meninges—Bibliography, 34;  
 April, p. xxvi; Bibliography,  
 42; Sept., p. xx.  
 Mental Diseases—Study of. . (455)  
 Mental Disturbance—Acute. . 515  
 Mental States in Epilepsy. . . \*389  
 Mental Torticollis. . . . . 514  
 Meralgia — Paresthetic — with  
 Traumatic Etiology . . . . . 280  
 Meralgia—Paresthetic. . 16, 56, 216

Metabolism—Myotonia—A Dis-  
 ease of. . . . . 612  
 Metastases in Fourth Ventricle  
 . . . . . 565  
 Migraine with Aphasia. . . . . 117  
 Migraine to Epilepsy—Relation  
 of. . . . . 118  
 Migraine—Internal Ophthalmo-  
 plegia in Ophthalmoplegic. . 230  
 Migraine—Ophthalmoplegic . . 215  
 Migraine — Relation Between  
 Trigeminal Neuralgia and. . \*129  
 Minor Epilepsy. . . . . 361  
 Miscellany. . . . . 128, 176, 238, 304  
 Modesty—Evolution of. . . . 446  
 Monocular Hysterical Amauro-  
 sis. . . . . \*444  
 Motor Neuroses—Bibliography,  
 34; April, p. xxvi; Bibliog-  
 raphy, 46; Nov., p. xx.  
 Motor Tracts. . . . . 57  
 Multiple Sclerosis—Differential  
 Diagnosis. . . . . 228  
 Multiple Sclerosis with Pro-  
 gressive Muscular Atrophy. . 106  
 Multiple Sclerosis, \*634; Bibli-  
 ography, 41; Sept., p. xviii.  
 Multiple Neuritis. . . . . \*577, 654  
 Multiple Neuritis with Atro-  
 phy. . . . . \*605  
 Multiple Neuritis and Beri-  
 Beri. . . . . \*645  
 Muscular Atrophy. . . . . \*274  
 Muscular Atrophy—Progressive  
 —of Myelopathic Origin. . . 299  
 Muscular Dystrophy, 47, 113,  
 \*347, 656; Bibliography, 36;  
 May, p. xxvi; Bibliography,  
 41; Sept., p. xviii.  
 Myelitis, 218; Bibliography, 39;  
 July, p. xx.  
 Myelitis—Hemorrhagic Ence-  
 phalitis and. . . . . 152  
 Myoclonus—Cases of. . . . . 562  
 Myoclonus—Epileptic. . . . . 525  
 Myopathy—Case of. . . . . 158  
 Myotonia—A Disease of Meta-  
 bolism. . . . . 612  
 Myxedema—Congenital. . . . . 15  
 Myxedema—Case of. . . . . 123  
 Myxedema—Bibliography, 31;  
 Mar., p. xx.  
 Narcolepsy. . . . . 363  
 Nasal Insufficiency—Hysterical. 410  
 Neuralgia—Epidemic. . . . . 225  
 Neuralgia—Paresthetic. . . . . 286  
 Neuralgias and Migraine—Re-  
 lation Between Trigeminal. . \*120



|  | PAGE          |  | PAGE                         |
|--|---------------|--|------------------------------|
| Neurasthenia—Bibliography, 47;<br>Dec., p. xviii.                              |               | Ophthalmoplegic Migraine .....                               | 215                          |
| Neuritis—Facial and Retrobul-<br>bar.....                                      | 150           | Optic Chiasm—Lesions of.....                                 | *135                         |
| Neuritis of Brachial Plexus....  | 649           | Pachymeningitis—Specific Cer-<br>vical.....                  | 281                          |
| Neuritis—Multiple.....   | *577, 654     | Palsy—Peripheral Facial .....                                | 553                          |
| Neuritis—Multiple—with At-<br>rophy.....                                       | *605          | Palsy—Remote Results in Ar-<br>tisan's.....                  | 140                          |
| Neuritis—Multiple—and Beri-<br>Beri.....                                       | *645          | Palsy—Treadler's .....                                       | (556)                        |
| Neuritis—Sub-Acute.....  | *603          | Paramyoclonus and Epilepsy                                   | 552, 561                     |
| Neurofibrosis—Generalized .....  | 415           | Paramyotonus.....  | 454                          |
| Neurotrophic Cutaneous Dis-<br>turbances.....                                  | 46            | Paranoia—Acute.....  | 546                          |
| Neurone—Anatomic—Cytologi-<br>cal Relationship to Nervous<br>Disease.....      | *469          | Paraplegia—Ataxic.....                                       | *577                         |
| Neurone Doctrine.....  | *506, *526    | Paraplegia—General Symptoms<br>of.....                       | 228                          |
| Neurone Doctrine—Relationship<br>to Nervous Disease.....                       | *469, *526    | Paraplegia—Syphilitic.....                                   | 162                          |
| Neurone—Pathological Changes<br>in Nervous Disease.....                        | *487          | Paralysis A g i t a n s—Bibliog-<br>raphy, 42; Sept., p. xx. |                              |
| Neurone—Size and Shape of<br>.....   | *526, (527)   | Paralysis—Double Amyotro-<br>phic.....                       | 429                          |
| Neurone—Bibliography, 39;<br>July, p. xx; Bibliography, 45;<br>Nov., p. xviii. |               | Paralysis—General.....                                       | 112                          |
| Neuroses—Etiologic Study of....  | 409           | Paralysis—Landry's—With Au-<br>topsy.....                    | *619                         |
| Neuroses—Functional—Occup-<br>ation in .....                                   | 364           | Paralysis—Landry's, *74, (90),<br>.....                      | *177, 434                    |
| Neurotic Atrophy—Primary,<br>.....   | 287, *324     | Paralysis of Peroneal Nerve in<br>Tabes.....                 | 230                          |
| Nerve Cell—Anatomical Physi-<br>ological and Pathological<br>Relations of..... | 219           | Paralysis—Pseudo—Hypertro-<br>phic.....                      | 664                          |
| Nerve—Fifth—Trophic Changes<br>in First Branch.....                            | 288           | Paralysis of Serratus Magnus.....                            | 215                          |
| Nerve Trauma—Bibliography,<br>43; Oct., p. xviii.                              |               | Paralysis—Syphilitic Spinal .....                            | 233                          |
| Nervous Disease—Anatomic—<br>Cytological Relationship to.....                  | *469          | Paralysis—Traumatic Periodic.....                            | 517                          |
| Nervous Disease and Neurone<br>Doctrine.....                                   | *469, *526    | Paralysis—Unilateral Progress-<br>ive Ascending.....         | *195                         |
| Nervous Disease—Pathological<br>Changes in Neurone in.....                     | *487          | Paralysis—Unusual Case of<br>Lead.....                       | 155                          |
| Nervous Disease—Use of Brain<br>Substance in.....                              | 615           | Paresis. See General Paresis.                                |                              |
| Nervous Equivalent of Fever.....   | 104           | Paresthetic Meralgia with Trau-<br>matic Etiology.....       | 289                          |
| Nervous People—Employment<br>for.....  | 364           | Paresthetic Meralgia.....                                    | 16, 56, 216                  |
| Nervous System—Gonococcus<br>Toxine—Its Action on.....                         | 230           | Paresthetic Neuralgia.....                                   | 286                          |
| New York Neurological Society<br>Transactions, 50, 106, 150, 212,<br>.....     | 281, 405, 652 | Parietal Convolution—Tumor<br>in.....                        | *244, (385)                  |
| Newspaper Science.....   | 208           | Parkinson's Disease—Cases of.....                            | 350                          |
| Occupation—Neuroses.....   | 364           | Pathological Changes in Neu-<br>rone in Nervous Disease..... | *487                         |
| Ophthalmoplegia in Ophthal-<br>moplegic Migraine—Internal.....                 | 230           | Pathology and Etiology of<br>Epilepsy.....                   | *394, 662                    |
|  |               | Pathology of Spinal Cord after<br>Embolus.....               | 414                          |
|  |               | Phantastica-Pseudologia—Ex-<br>ample of.....                 | 463                          |
|  |               | Philadelphia Neurological So-<br>ciety.....                  | 113, 158, 287, 349, 453, 553 |
|  |               | Physiology—Bibliography, 34;<br>April, p. xxvi.              |                              |
|  |               | Plantar Reflex—Study of.....                                 | *305                         |
|  |               | Polio-myelitis—Bibliography, 36;<br>May, p. xxvi.            |                              |

|   | PAGE        |   | PAGE          |
|---|-------------|---|---------------|
| Poliomyelitis—Acute.....  | 116, *205   | Regurgitation.....  | 443           |
| Poliomyelitis—Etiology of Acute Anterior.....   | 227         | Retrograde Degeneration.....  | 614           |
| Polyneuritis—Painful Tubercular.....  | 446         | Retropulsive Epilepsy.....  | 204           |
| Pregnancy and Epilepsy.....   | 517         | Rigid Spine—A Case of.....  | 52            |
| Progressive Ascending Paralysis—Unilateral.....   | *195        | Rigidity—Ankylotic Spinal.....  | *1            |
| Progressive Amyotrophy of Traumatic Origin.....   | 271         | Rigidity of Spine.....  | *542          |
| Progressive Muscular Atrophy.....   | 106, 290    | Rheumatism—Cerebral.....  | 361           |
| Progressive Muscular Dystrophy.....   | *547, 656   | Rhizomelic Spondylosis.....   | 291, 558      |
| Prognosis in Epilepsy.....  | *390        | Rhizomelic Spondylosis—Bibliography, 48; Dec., p. xx.                   |               |
| Pruritus and Trichotillomania in General Paresis.....   | 55          | Rumination.....   | 443           |
| Pseudo-Hypertrophic Paralysis.....  | 664         | Sarcoma of Third Ventricle—Metastases in Fourth.....                    | 565           |
| Pseudologia Phantastica—Example of.....   | 463         | Sarcomatosis—Diffuse.....   | 169           |
| Pseudoparalysis—Syphilitic.....   | 157         | Scapulo-Humeral Reflex.....   | 404           |
| Pseudo-Science—Christian—and Psychiatry.....  | 438, (438)  | Science—Newspaper.....  | 208           |
| Psychical Effects of Alcohol—Investigations on.....   | 330         | Sclerosis—Amyotrophic Lateral.....                                      | 165, 213, 556 |
| Psychiatry—Christian Pseudo-Science and.....  | 438, (438)  | Sclerosis—Differential Diagnosis of Multiple.....                       | 228           |
| Psychiatry—Etiology—Bibliography, 30; Feb., p. xx; Bibliography, 32; Mar., p. xxvi; Bibliography, 40; Aug., p. xx.                                    |             | Sclerosis Ischiadica.....   | 358           |
| Psychiatry—Treatment—Bibliography, 30; Feb., p. xx; Bibliography, 32; Mar., p. xxvi; Bibliography, 33; April, p. xx; Bibliography, 38; June, p. xxvi. |             | Sclerosis—Multiple.....   | 106, *634     |
| Psychological Atomism.....  | 211         | Segmentary Angioma.....   | 466           |
| Psychosis—Acute.....  | *41, (41)   | Senile Epilepsy.....  | 560           |
| Pupillary Reflexes.....   | 123         | Sensibility—Correlative Variation of.....                               | 465           |
| Pupillary Reflexes—Report on.....   | 200         | Sensory Anesthesia—Pathological Findings in Case of Cutaneous.....      | 351           |
| Pyramidal Cells—Changes in Large.....   | 73          | Sensory Neuroses—Bibliography, 48; Dec., p. xx.                         |               |
| Pyramidal Giant Cells—Observation on Changes of.....  | 486         | Spinal Commotions—Cerebral and.....                                     | 173           |
| Raynaud's Disease.....  | 660         | Sleep—Prolonged—Case of.....  | 666           |
| Raynaud's Disease and Acromegaly.....   | 463         | Spinal Cord after Embolus—Pathology of.....                             | 414           |
| Raynaud's Disease in the Insane.....  | *272        | Spinal Cord Injuries from Fracture of Vertebrae.....                    | 614           |
| Reflex—Femoral.....   | 297         | Spinal Cord—Injury.....   | 113           |
| Reflex—Plantar—Study of.....  | *305        | Spinal Cord—Tumor of.....   | *580          |
| Reflex—Scapulo—Humeral.....   | 404         | Spinal Cord Tumors—Bibliography, 42; Sept., p. xx.                      |               |
| Reflexes—Clinical Study of.....   | *375, (381) | Spinal Paralysis—Syphilitic.....  | 233           |
| Reflexes—Pupillary.....   | 123         | Spinal Cord—Researches on Failure of Knee-Jerk in Lesions of Upper..... | 296           |
| Reflexes—Report on Pupillary.....   | 200         | Spinal Cord Trauma.....   | *65           |
|   |             | Bibliography, 48; Dec., p. xx.  |               |
|   |             | Spinal Membranes—Diagnosis—Surgical Treatment of Tumors of.....         | 226           |
|   |             | Spinal Nerves—Bibliography, 46; Nov., p. xx.                            |               |
|   |             | Spinal Rigidity—Ankylotic.....  | *1            |
|   |             | Spine—Rigidity of.....  | *542          |
|   |             | Spine—Rigid.....  | 52            |
|   |             | Spondylosis—Rhizomelic.....   | 291, 558      |
|   |             | Status Epilepticus.....   | *387          |
|   |             | Studies in Astereognosis.....   | *569          |

PAGE

PAGE

|  |          |   |             |
|--|----------|---|-------------|
| Sub-Acute Pressure—Neuritis.....                             | 603      | Treatment of Epilepsy,*391, 566, 615                        |             |
| Subarachnoidal Injections .....                              | 365      | Treatment of Epilepsy—Surgical .....                        | *394        |
| Sulphocarbolate of Soda in Chorea.....                       | 234      | Treatment—Occupation—in Functional Neuroses .....           | 364         |
| Surgical Treatment of Epilepsy.....                          | *394     | Treatment of Pain in Tabes Dorsalis .....                   | 366         |
| Sympathetic Chain—Disease of Thoracic Division of.....       | 282      | Treatment—Torticollis and its.....                          | 284         |
| Symptomatology — Bibliography, 46; Nov., p. xx.              |          | Treatment of Trophic Lesions of Foot .....                  | 194         |
| Syndrome—Double—of Weber with Autopsy.....                   | 466      | Treatment—Tumors of Spinal Membranes—Surgical .....         | 226         |
| Syringomyelia .....  | 150      | Tremor—Atypical Case of.....                                | 290         |
| Bibliography, 37; June, p. xx.                               |          | Trephining for Traumatic Epilepsy .....                     | 566         |
| Systemic Disease of Lateral Columns.....                     | 541      | Trichotillomania and Pruritus in General Paresis.....       | 55          |
| Syphilis—Bibliography, 37; June, p. xx.                      |          | Trigeminal Neuralgias and Migraine—Relation Between.....    | *129        |
| Syphilis of Brain.....                                       | 665      | Tropho-Edema—Chronic Hereditary .....                       | 296         |
| Syphilitic Paraplegia.....                                   | 162      | Trophic Lesions of Foot—Treatment of.....                   | 194         |
| Syphilitic Pseudoparalysis .....                             | 157      | Tubercular Polyneuritis—Painful .....                       | 446         |
| Syphilitic Spinal Paralysis.....                             | 233      | Tumor—Brain .....   | *103, 172   |
| Tabes Dorsalis—Cases of.....                                 | 349      | Tumor—Brain—Differential Diagnosis .....                    | 457         |
| Tabes Dorsalis—Treatment of Pain in.....                     | 366      | Tumor—Brain—General Symptoms of .....                       | 457         |
| Tabes and General Paralysis.....                             | 112      | Tumor—Brain—Value of Eye and Ear Symptoms in.....           | 460         |
| Tabes—Paralysis of Peroneal Nerve in.....                    | 230      | Tumor of Cerebellum.....                                    | 151         |
| Tabes and Traumatism.....                                    | 225      | Tumor Compressing the Cauda Equina.....                     | 108         |
| Tabetic Arthropathies and Radiography .....                  | 564      | Tumor—Congenital—of Lumbar Region.....                      | 374         |
| Tabetic Symptoms and Exaggerated Knee-Jerks — Case with..... | 159      | Tumor—Intracranial .....                                    | 216         |
| Technic—Bibliography, 43; Oct. p. xviii.                     |          | Tumor — Pathological Report of.....                         | *263        |
| Telangiectasia with Unilateral Hypertrophy .....             | 297      | Tumor in Parietal Convolution .....                         | *244, (385) |
| Temperature in Apoplexy.....                                 | 226      | Tumor of Spinal Cord.....                                   | *580        |
| Tetanus—Bibliography, 30; Feb., p. xxvi.                     |          | Tumors of Spinal Membranes —Surgical Treatment .....        | 226         |
| Tetanus—Traumatic.....                                       | 516      | Tympanites — Abdominal — of Hysterical Origin .....         | 358         |
| Thyroidectomy for Graves' Disease .....                      | 405      | Unilateral Hypertrophy with Telangiectasia.....             | 297         |
| Tic of Foot—Cases of.....                                    | 518      | Unilateral Internal Hydrocephalus .....                     | 293         |
| Torticollis—Mental. ....                                     | 514      | Vasomotor Functions — Disturbances of.....                  | 271         |
| Torticollis and Its Treatment.....                           | 284      | Vision—Curious Hysterical Disturbances of.....              | 285         |
| Toxics and Auto-Toxics in Production of Epilepsy.....        | *338     | Visual Tracts—Brain from Case of Congenital Absence of..... | 357         |
| Trauma of Spinal Cord.....                                   | *65, 614 | Weber—Double Syndrome of.....                               | 466         |
| Traumatic Epilepsy — Trephining for .....                    | 566      | Word-Deafness .....   | 406         |
| Traumatic Origin — Progressive Amyotrophy of.....            | 271      |   |             |
| Traumatic Periodic Paralysis.....                            | 517      |   |             |
| Traumatism—Tabes and .....                                   | 225      |   |             |
| Treatment of Chorea by Sulphocarbolate of Soda.....          | 234      |   |             |

## INDEX TO BOOK REVIEWS.

|  | PAGE |
|--|------|
| Barker, L. F., The Nervous System and its Constituent Neurons.....   | 174  |
| Binswanger, O., Die Epilepsie.....   | 235  |
| Corwin, A. M., Essentials of the Physical Diagnosis of the Thorax.....   | 126  |
| de Sanctis, Sante, I Sogni: Studi Psicologici e Clinici di un Alienista.....   | 60   |
| Eulenburg, A., Krankheiten der Peripherischen Nerven.....  | 368  |
| Flatau, E., and Jacobsohn, L., Handbuch der Anatomie und Vergleichenden Anatomie des Central Nerven Systems der Säugethiere.....                         | 61   |
| Flatau, E., and Jacobsohn, L., Jahresbericht über die Leistungen und Fortschritte auf dem Gebiete der Neurologie und Psychiatrie.....                    | 304  |
| Gordinier, H. C., A Gross and Minute Anatomy of the Central Nervous System.....  | 367  |
| Hinshelwood, J., Letter, Word and Mind Blindness.....  | 303  |
| Kirkpatrick, T., Raynaud's Disease. Its History, Causes, Symptoms, Morbid Relations, Pathology and Treatment.....  | 417  |
| Lehr, M., Die Nervösen Krankheitserscheinungen der Lepra, mit Besonderer Berücksichtigung ihrer Differential-Diagnose.....                               | 124  |
| Letchworth, W. P., Care and Treatment of Epileptics.....   | 617  |
| Moore, W. E., Orthopedic Surgery.....  | 303  |
| Nancrede, C. B., Essentials of Anatomy.....  | 127  |
| Obersteiner, H., and Redlich, E., Krankheiten des Rückenmarkes.....  | 126  |
| Penrose, C. B., A Text-Book of Diseases of Women.....  | 418  |
| Potts, C. S., Lea's Series of Pocket Text-Books. Nervous and Mental Diseases. A Manual for Students and Practitioners.....                               | 368  |
| Pryor, W. R., Treatment of Pelvic Inflammation Through the Vagina.....   | 304  |
| Roux, J. C., Les Lésions du Systeme Grand Sympathique dans le Tabes, et leur Rapport avec les Troubles de la Sensibilité Viscerale.....                  | 567  |
| Sachs, H., and Freund, C. S., Die Erkrankungen des Nerven Systems nach Unfällen, mit Besonderer Berücksichtigung der Untersuchung und Begutachtung.....  | 467  |
| Saunders' American Year Book of Medicine and Surgery.....  | 128  |
| Savill, T. D., Clinical Lectures on Neurasthenia.....  | 124  |
| Stelwagon, H. W., Essentials of Diseases of the Skin, Including the Syphilodermata.....  | 61   |
| Thomson, St. C., The Cerebrospinal Fluid. Its Spontaneous Escape from the Nose. With Observations on its Position and Function in the Human Subject..... | 125  |
| Thorndike, E. L., Animal Intelligence: An Experimental Study of the Associate Processes in Animals.....  | 60   |
| van Gehuchten, H., Anatomie du Systeme Nerveux de L'Homme.....   | 174  |
| Warren, J. C., The International Text-Book of Surgery by American and British Authors.....   | 416  |
| Ziehen, T., Makroskopische und Mikroskopische Anatomie des Rückenmarks.....  | 127  |

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THE  
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**Original Articles.**

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PROGRESSIVE ANKYLOTIC RIGIDITY OF THE SPINE  
(SPONDYLOSE RHIZOMÉLIQUE).\*

By B. SACHS, M.D., and J. FRAENKEL, M.D.

Chronic rigidity of the spine has been observed often enough by neurologists and orthopedic surgeons. The condition has commonly been associated in their minds with arthritis deformans, or articular rheumatism. Little attention was paid to the particular significance of this spinal deformity until within the last few years. Short as this discussion has been, it has brought to light the fact that every conceivable form of chronic vertebral disease has been included in the newly described type. It will be well, therefore, to endeavor to summarize the results of recent studies, to give the impressions gained from our own experience, and to state the special points that need further elucidation.

It would appear that in 1892 and 1893 von Bechterew<sup>1</sup> published a paper, entitled "Rigidity and curvature of the vertebral column as a special form of disease." Further consideration of the subject was delayed until 1897, when the same author wrote another paper,<sup>2</sup> entitled "On ankylosis or rigidity of the vertebral column." The symptoms of this condition were:

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\*Read before the New York Neurological Society, Nov. 7, 1899.

The article by Zenner in the November number of this Journal reached the authors of this paper too late to be considered by them.

<sup>1</sup> von Bechterew, *Wratsch*, 1892, No. 36; also *Neurologisches Centralblatt*, 1893, p. 426.

<sup>2</sup> v. Bechterew, *Deutsche Zeitschrift für Nervenheilkunde*, Vol. XI, p. 327.

First, immobility, complete or partial of the whole or part of the vertebral column, without any distinct painfulness on percussion. Secondly, a kyphosis of the cervical portion of the vertebral column, the head being bent forward and downward. Thirdly, a paretic condition of the muscles of the trunk, the neck and the extremities, with slight atrophy of the muscles of the back and of the shoulder girdle. Fourthly, a diminution of sensation within the distribution of the cutaneous branches of the nerves supplying the back and of the lower cervical plexus. Fifthly, paresthesiæ and pain in the back and in the neck, in the extremities and in the vertebral column. Occasionally symptoms occur pointing to irritation of the motor nerves as well. All other joints remain exempt, in spite of the progressive character of the disease. Traumatism and hereditary influences are the chief etiological factors. Briefly then, von Bechterew's cases were characterized by a chronic rigidity of the vertebral column, plus root symptoms, but without the involvement of any other joint.

In the same volume of the *Zeitschrift für Nervenheilkunde* in which von Bechterew's article appeared, Struempell<sup>3</sup> published a short communication, entitled "Observations on a form of chronic inflammation of the vertebral column and of the hip joint, with ankylosis." He points very properly to a statement made in the first edition of his text book (1884) in the chapter on chronic articular rheumatism and arthritis deformans. In the American edition, p, 861, we read: "A remarkable and, as it seems to us, unique disorder may be mentioned in passing. It leads very gradually and painlessly to a complete ankylosis of the entire spinal column and the hip joints, so that head, trunk and thighs are firmly united and completely stiffened, while all the other joints retain their normal mobility. It need scarcely be said that this necessarily causes a peculiar modification of the carriage and gait of the sufferer. We have seen two cases of this peculiar affection which resembled each other very closely."

A third case is described in the recent article. There is a general resemblance between the cases referred to by Struempell and the type established by von Bechterew; the chief differ-

<sup>3</sup> Struempell. *Deutsche Zeitschrift für Nervenheilkunde*, Vol. XI, p. 338.

ence between the two consisting in the implication of the hip joints and the entire absence of spinal root symptoms in the type described by Struempell. In the following year, 1898, Marie and Astie<sup>4</sup> took up the same subject, reporting cases very much like those described by Struempell, and proposing for these the name "spondylose rhizomélique." Marie's title was to imply the simultaneous involvement of the vertebral joints and of the hip or shoulder joints (root joints); but, inasmuch as other joints have been involved in some of the cases since reported, there is some justice in von Bechterew's<sup>5</sup> criticism that the name proposed by Marie is not wholly correct.

In addition to the leading articles just mentioned, a number of clinical contributions have been made by Baeumler,<sup>6</sup> Gowers,<sup>7</sup> Oppenheim,<sup>8</sup> Hoffmann,<sup>9</sup> of Duesseldorf; Valentini,<sup>10</sup> Bregman,<sup>11</sup> Sænger,<sup>12</sup> Schataloff,<sup>13</sup> Mutterer,<sup>14</sup> Hoffa,<sup>15</sup> Popoff,<sup>16</sup> and Leri.<sup>17</sup> Some of these were published prior to the recent articles of Struempell and of Marie. It should be especially noted that in the first edition of his book (1894) Oppenheim has a short section on "Arthritis deformans of the vertebral column," in which he refers to a group of cases bearing all the characteristic symptoms of von Bechterew's type. A careful reading of all of these reports makes it certain that there is a general resemblance between the cases described by these various writers. In some there is a superficial resem-

<sup>4</sup> Marie and Astie, *Presse Médicale*, No. 82 October, 1898; also *Revue de Médecine*, 1898.

<sup>5</sup> v. Bechterew, *Deutsche Zeitschrift für Nervenheilkunde*, Vol. XV, p. 37.

<sup>6</sup> Baeumler, *Deutsche Zeitschrift für Nervenheilkunde*, Vol. XII.

<sup>7</sup> Gowers, "Manual of Diseases of the Nervous System," 2d Edition, Vol. I, p. 263.

<sup>8</sup> Oppenheim, "Lehrbuch der Nervenkrankheiten," 2d Edition, p. 228.

<sup>9</sup> Hoffmann, *Deutsche Zeitschrift f. Nervenheilkunde*, Vol. XV, 1899, p. 28.

<sup>10</sup> Valentini, *Deutsche Zeitschrift f. Nervenheilkunde*, Vol. XV, p. 239.

<sup>11</sup> Bregman, *Deutsche Zeitschrift f. Nervenheilkunde*, Vol. XV, p. 250.

<sup>12</sup> Sænger, *Neurologisches Centralblatt*, 1898, p. 1144 (Society report).

<sup>13</sup> Schataloff, *Neurolog. Centralblatt*, 1898, p. 828 (Society report).

<sup>14</sup> Mutterer, *Deutsche Zeitschrift für Nervenheilkunde*, Vol. XIV, p. 144.

<sup>15</sup> Hoffa, *Sammlung klinischer Vortraege (Volkman)*, No. 247, 1899.

<sup>16</sup> Popoff, *Neurologisches Centralblatt*, 1899, p. 294.

<sup>17</sup> Leri, *Revue de Médecine*, No. 8, 1899.

blance, at least, to an arthritis deformans, which has by chance involved the vertebral column, and in others, as, for instance, in the one recently reported by Hoffmann, the condition is evidently a chronic rheumatic affection of the vertebral joints. Von Bechterew,<sup>18</sup> with a natural desire to claim priority in this matter, insists that his and the Struempell-Marie type have little in common, except the rigidity of the spine. But are the differences so striking that distinct types of vertebral disease should be maintained? A little light has been thrown on this discussion by the results of post-mortem examination.

V. Bechterew, who on clinical grounds believed that the process which caused the ankylosis of the vertebral column also involved the spinal nerve roots, found some proof of this assertion in a post-mortem examination<sup>18</sup> which he was enabled to make on one of the first patients he described. In this instance he found a chronic leptomeningitis of the upper cervical region compressing the spinal nerve roots. The posterior roots, particularly in the cervical region, were degenerated; the motor roots were less intensely affected. In the cervical and upper dorsal segments there was a marked degeneration of the posterior columns, more especially of the column of Goll and of a portion of the column of Burdach. These were secondary to the root degeneration; the gray matter of the cord had not been diseased. The degeneration of the spinal nerve roots was supposed to be due to the changes in the cells of the spinal ganglia; the spinal ganglia being adherent to the dura mater. The surprise of this post-mortem examination was the discovery that there was no primary lesion of the vertebræ or of the joints, and the author supposed that the rigidity of the vertebral column was secondary to a parietic condition of the muscles, resulting from the compression of the nerve roots, resembling in this respect the changes found in the vertebral column, in syringomyelia, and some other spinal cord affections.

The interpretation given by von Bechterew may be applicable to this special case in which, by the way, the root symptoms were much more marked than in any other patients

<sup>18</sup> v. Bechterew, *Deutsche Zeitschrift f. Nervenheilkunde*, Vol. XV, p. 45.



thus far described. Strong corroboration of von Bechterew's views is to be found in the fact that the spinal root pains preceded by many years the appearance of the vertebral ankylosis. If there were any doubt as to the identity of the two types of chronic spinal rigidity, these doubts would be confirmed by the results of the pathological examinations made by Marie and Leri. The former based his opinion of the pathology of the disease upon the study of a skeleton in the Musée Dupuytren, in which he found marked proliferation of the lumbar vertebræ and of the bones entering into the hip joint. Ossification had taken place between the various vertebræ. Marie inferred that the ossification of the anterior longitudinal ligament and of the ligaments connecting the ribs with the vertebræ must have caused the complete immobility of the spine.

In the *Revue de Médecine* of this year Leri<sup>17</sup> describes the autopsy on a case of "spondylose rhizomélique" recognized as such during life, in which there was complete ossification of the ligaments and an hypertrophy and ankylosis in the joints of the extremities. The ossification in the vertebral column was most marked on the convexity of the vertebral bodies. The autopsical findings of von Bechterew and of Leri differ to such a marked degree that if the findings of each are to stand as the anatomical substrata of the respective types, the two must be regarded as widely differing morbid conditions; but we repeat that von Bechterew's case was an exceptional one in many respects, and further post-mortem examination must be awaited before any positive conclusions can be reached. Meanwhile, it will be well to consider the various types from a clinical standpoint. But before doing so, let us present once more the salient features of each group of cases.

*Type of von Bechterew:*

Chronic rigidity of spine, often limited to cervical region; other joints free. Root symptoms predominate. Anatomical findings: chronic leptomeningitis with root and spinal cord changes; vertebral joints not affected.

*Type of Strumpell-Marie:*

Chronic rigidity of the spine with involvement of shoulder or hip joints. No root symptoms. Anatomical findings: ossification of ligaments, hypertrophy and ankylosis of joints.

*Case 1.*\*—B. B., 37 years of age, a Russian, tailor by trade; married; father of two living children; has lost none, and wife has had no miscarriages. He has been in this country 13 years. His parents died at advanced age; has four brothers, and has not lost either brothers or sisters. In the family there is no history of rheumatism, nor of tuberculosis; nor is there any neuropathic taint. In his early infancy he was healthy and had no illness, until the age of ten years, at which time he had an attack of influenza. Denies venereal infection. Habits have been good, drinking and smoking in moderation. Has been a hard worker; has never been exposed to so-called rheumatic influences. Had no deformity in former years; walked erect; was examined 15 years ago by an army physician and found fit for service. The present illness began about six years ago. The only cause that he can assign for it is a "cold." The disease began with pains in the left knee upon rising. This pain was increased on the attempt to walk. He is very certain that he had no fever and that there was no swelling or redness about the knee. The pain was worse during the day and continued with intermission for a year and a half, when he entered the Presbyterian Hospital. At this time the leg was put into some sort of extension apparatus. After two months he was considerably improved and was able to walk for one year without experiencing much pain. At the end of this period the pains appeared again in the same place, and, after lingering along for a few months, he entered the Montefiore Home.

The patient states that a slight cough accompanied the onset of the present illness. The expectoration was never bloody, and there were no night sweats or fever. One of the former physicians of the Home made the diagnosis of pulmonary phthisis and tubercular coxitis. The cough continued during the first year and a half of his stay at the Home, but he never had any fever, or any of the other signs or phthisical affections, and repeated examinations of the sputum failed to reveal the presence of bacilli. The orthopedic surgeons of the Home inclined to the diagnosis of tubercular coxitis. At the Home the knee was again put into an extension apparatus for two and a half months, but the patient's condition became distinctly worse. Both knees now became stiff; the

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\* After sending this paper to press we note that this patient's condition has also been fully described by Dr. Dana, (*Medical News*, Nov. 25, 1899.) His and our articles were prepared independently of one another; it is gratifying to note that the conclusions reached in both are practically the same.

The patients designated as cases I, III and IV were exhibited at the meeting.

left was swollen a little at times and was painful when manipulated, but was never red. He was kept in bed for ten months, during which time the hips and the lower part of the spine became rigid. The patient did not, however, complain of great pain in either region. The rigidity of the hips and spine became progressively worse, until he reached the present condition. At no time was there a rise of temperature. From inspection of the patient, it is evident that he is stiff in the knees, hips and spine, all other joints remaining exempt. His bowels are regular. Sleep is disturbed, awakening often during the night; micturition normal; appetite poor; has no pain nor ache, and according to his own statement, if it were not for the stiffness of the back he would feel well.

The examination of the patient in his present condition reveals a firm pulse of 96; respiration 18; weight 82 pounds; temperature normal. Patient is of medium height, but generally emaciated. When asked to mount the scales he cannot do so, though they are only four inches from the ground. He is compelled to use two supports and raise entire body the required distance. He is not able to walk or to stand without being held, both lower extremities being entirely rigid, the left one adducted and rotated inwards; the ball of the foot only touching the floor. The left leg forms an angle of about  $45^{\circ}$  with the right. The axes of both thighs are parallel and very close together, especially at the knees. In attempting to walk he is compelled to move the extremities in a rotary manner, makes short steps, shoves feet forward and jerks the trunk around. The spine is rigid, patient bending forward, the shoulders distinctly stooping, the left higher than the right. This forward tendency of the head and of the upper thorax is so marked that when he stands erect the left shoulder is on a level with the malar bones. The entire body is inclined toward the left, thus enabling him, while standing, to rest on the left foot. When standing erect only the toes of the left foot are in contact with the floor.

... He cannot sit in an ordinary wheeling chair, but must rest on a sloping surface. When in bed, he turns as though made of a solid piece, and using his upper extremities only. The head is brachycephalic, and there is distinct occipital depression. The pupils are equal, the reaction normal, and all ocular and facial movements are normal. The pharyngeal reflex is present. All jaw movements, vertical and lateral, can be performed properly. Shoulder movements are free and limited to the left. Nodding movements are made imperfectly, and flexion, as well as extension movements of the head

are also limited. The upper extremities are moved freely. The muscles are flabby, the reflexes cannot be elicited; myotatic irritability is diminished; there is no diminution of the muscular power of the upper extremities. The thorax is flattened, especially in its upper portions. The sternum is very prominent. There is retraction of both apices, with prominent clavicles. There is a higher percussion note over both apices. There is somewhat diminished breathing over both lungs. No râles are heard, but inspiration is rather sharp with prolonged expiration. On deep inspiration the circumference of the chest is  $76\frac{1}{2}$  c. m., an increase of only  $\frac{1}{2}$  c. m. over the normal circumference. Heart conditions are entirely normal. Liver and spleen not enlarged. Evident rigidity of the abdominal muscles. The abdominal aorta can be easily palpated and appears to be near the surface. The knee-jerk is lively on the right side, normal on the left, and ankle-clonus can be elicited at times. The Achilles tendon reflex cannot be elicited on either side; plantar reflexes lively—flexion of toes and dorsal flexion of foot. Increased myotatic irritability. There is distinct atrophy of the extensors of the thighs and legs, more especially on the left side. No marked changes in electrical reaction. Motion below the ankles appears to be fairly normal; all the other joints are fixed. There is no sensory disturbance. A globular swelling of both knees; no tenderness; no redness. The joints appear to be hard and bony. As for the spinal column, it is seen that the vertebræ are rigid, that the dorsal processes have a marked curvature, and the lower are especially prominent. There is marked prominence of upper lumbar vertebræ; distinct depression of the lower two, while the sacrum is unusually prominent. There is no tenderness either over the spinous processes or along the nerve trunks. No spiculæ of bone are to be found on the dorsal surface or on palpation through the pharynx. A small amount of albumin has been found in the urine, but casts have never been detected. The specific gravity is 1020; there is no sugar; the reaction is acid, and of urea we find 7 grains to the ounce.

Under the advice of Dr. Elliott, the patient was anesthetized, and during narcosis all affected joints remained absolutely rigid. It is worth adding that there was an apparent persistent ankle-clonus during the anesthesia. In spite of all treatment, the condition has been growing steadily worse.

It will be seen from the history of the above case that the condition corresponds in almost every essential feature with the type of Struempell-Marie. The only exceptional circum-

stance to be noted is that the affection began in the knee and appeared to be limited to it for a considerable period of time before it reached the spine. It is true of all the cases thus far reported of this type that they affect men and women\* in middle life; that the condition is developed in a subacute or chronic fashion, and that the stiffness of the hip and of the vertebral joints, occasionally of the knee joints, terminating in complete ankylosis, constitute the sole diagnostic features of the disease. The absence of root symptoms is entirely characteristic. With this first case let us compare the condition of another patient who has been under the observation of Dr. Fraenkel.

*Case 2.* J. S., 53 years of age, Irish, painter. Is married. Family history, in spite of thorough questioning, is negative. Parents lived to an old age. Two brothers are living and well. No indication of rheumatism, tuberculosis or other degenerative taint in family. Patient is married 25 years and has 4 living and healthy children; there were no miscarriages. Patient was healthy in infancy and childhood; was moderate in his habits; had no exposures or severe colds. Thirty-two years ago he had a chancre and was treated by internal and external medication, but claims never to have had any other manifestation of the disease. Otherwise he was perfectly well up to the onset of present disease, excepting for the fact that he was often subject to "sore throats." And he states that his present disease was the immediate consequence of such an attack of sore throat, which the then attending physician called "a mild attack of diphtheria." This happened 18 years ago. Since then he has been having sore throats on and off, but less often than formerly.

The first symptoms of the present illness were "rheumatic pains" all over for about two weeks, and then all settled in the hips. At no time was there fever, swelling or pain in the last named joints, but slowly progressive interference with the excursions of these joints and the immediately neighboring parts of the vertebral column was the constant and only symptom. In all other respects the patient believed himself thoroughly well. He was a man of average height, of good bony and muscular development; well nourished, and shows no signs of his age—hair being colored and skin remarkably elastic. Vegetative organs and circulatory system normal;

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\*It is evident from the communications of Hoffa and of Collins (at the meeting) that women also are subject to the disease.

no evidence of arterial atheroma. Head is freely movable in all directions; there is no evidence of involvement of any of the joints of the cervical part of the vertebral column. Shoulder, elbow, wrist and finger joints absolutely free. Respiratory excursions of the thorax are normal. Expansion  $1\frac{1}{2}$  inches. The vertebral column is not tender to percussion, pressure or jarring. The lumbar part of the column, however, is somewhat more prominent, and the musculature here feels somewhat contracted. The excursions of the lower dorsal and lumbar part of the column are almost absolutely abolished. The hip joints are entirely ankylosed; passive movements elicit no cracking or pain. The knee, ankle and toe joints are free. The spincters are normal.

There is no evidence of muscular atrophy anywhere. Sensibility and reflexes normal. The gait is very peculiar. Evidently the lower part of the column and the hip are firmly soldered, and patient, therefore, manages to go about in a rotary fashion, not lifting the feet from the floor, but by rotating them alternately along the vertical axis and bending the legs slightly in the knees. In a similar way the patient is unable to look sideways without turning around altogether. In bending, he does so carefully by letting himself down on the arm and bending his knees. He sits down in the same fashion. Palpation of vertebral column from pharynx and along the spinous processes is negative. No osseous spiculæ evident.

In this case we may readily see the close resemblance to the Struempell-Marie type.

*Case 3.* H. B., also a Russian, 48 years of age, tailor; is father of five living children, and has been in this country 11 years. A brother of his died of a disease characterized by weakness of the legs. Patient never smoked, was very moderate in drinking, and denies venereal infection. As a boy had morning headaches for one year. At the age of 15 years had a fever which kept him in bed for two weeks; lost his hair and became bald. Alopecia is probably due to favus; turned gray very early in life. His present illness began two and a half years ago, with pains in the sacral and lumbar regions. There is no history of trauma and no other disease preceded the onset of the present illness. The legs began to tremble and when he attempted to rise the heels began to beat a tattoo on the floor, and there was a drawing up of all the extremities. Six years ago he had pains in the right shoulder and forearm, lasting one year. There was no fever, but the affection was supposed to be rheumatic. The shoulder was somewhat swollen, and he could not use the hand and carried

it in a sling. The right leg was worse. In a few months he began to walk badly. He was bent forward, the spine forming an angle in the lumbar region. There was pain in this region on pressure. Difficulty in micturition was not pronounced, but there was chronic constipation and the sexual function was diminished. The trembling of the legs subsided, but the pain in the back became worse. The following is the condition of the patient as noted in the history of a year ago at the Montefiore Home.

The man is tall, well nourished, abundant subcutaneous fat. Hair gray, marked alopecia. The ears are asymmetrical, has a torus palatinus. When standing the right shoulder is higher than the left. He rises with difficulty and exhibits jerking movements of the muscles of the thighs. Upon sitting down he drops heavily into the chair, falling, as it were, through the last few inches of space. He bends forward and to the left, and has a spasto-paretic gait. The right leg is held stiffer than the left and is dragged more distinctly. The patient thinks that the peculiarity of the gait is due to pains in the back. The ocular movements, the heart and also the viscera are entirely normal. The tendon reflexes of the upper extremity are lively. Slight diminution of the gross muscular power of the right lower extremity. He cannot raise the right foot as well as he can the left. Upon letting the right leg down it drops the last few inches very heavily. The knee-jerks are lively and there is ankle-clonus on both sides. The plantar reflex is diminished on the left side. Sensation is normal throughout the body. There is distinct tenderness of the 12th dorsal vertebra, and this increases as we go downwards. There is some tenderness in the 4th left intercostal space. The left part of the vertebral column is distinctly rigid, and the muscles are in a condition of spastic contracture. During an examination made by Dr. Fraenkel, January 14, 1899, it was noted that there was a slight paralysis of the right lower extremity, scarcely any on the left side. The abdominal and cremasteric reflexes were lively on both sides, all other reflexes as at the time of first examination. Sensation did not appear to be altered, except that it was slightly diminished in the tips of the toes. Deep sensibility was normal.

On March 2, 1899, it was noted that the patient complained of having become much worse. He felt the pain, in the form of a semi-girdle sensation, radiating to the buttocks, and it is much more severe and more constant. He also feels that the right lower extremity is more contracted, and in both lower extremities there are constant and annoying paraesthesiae. When patient attempts to stand erect the whole body is curved.

He leans over to the left side, so that the umbilicus and jugular notch are not in a straight line. On the posterior aspect of the trunk there was a distinct curvature of the right ribs, and the lower portion of the vertebral column was twisted. He drags the right lower extremity and throws the weight of the body on the left. There is no ataxy, and resistance to passive movements is much less than on previous examinations. There is, however, very little actual paralysis. The tendon jerks in the upper extremities are lively; abdominal reflexes are lively on the left side and very much diminished on the right. The cremasteric is lively on the left and hardly present on the right. The plantar reflex is lively on the left and diminished on the right. The knee-jerk is lively on the right side, absent on the left; but a tapping of the left tendon is followed by contracture of the right side in all positions. The right Achilles tendon jerk is lively. No ankle-clonus. Left Achilles tendon jerk present, but much weaker than right. The tactile sense was preserved, but slightly diminished in peripheral parts of both extremities. Pain sense similarly affected, with spots of analgesia over peripheral parts. Around upper abdomen a girdle of hyperaesthesia. Lower vertebral column is carried stiffly and is tender to percussion from tenth dorsal spine downwards. Measurements carefully made showed a slight atrophy of right thigh and hip. Faradic and galvanic stimulation normal.

In view of the sudden development of increased root symptoms, of the increase in pain and of the loss of the left knee-jerk, the possibility of a neoplasm or of an exudate compressing the nerve roots was considered, and the patient was referred to Dr. Gerster, at the Mt. Sinai Hospital, for an exploratory operation. A laminectomy was done, two laminae (D. XI and XII) were removed, and a portion of the enormously thickened lining was excised. No tumor or exudate was found; during the operation the surgeon remarked upon the unusual thickness of the laminae and of the lining membrane.\* Sections of this membrane revealed a marked increase of connective tissue. The patient made a good recovery; the rigidity is distinctly improved, and the knee-jerk has returned. His walk and his pains have been improved.

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\*In the hospital records the surgeons stated "the periosteum and dura were greatly thickened."



Were it not for the development of the symptoms chiefly in the lumbar region, although the shoulder joint was also affected during an early period of the disease, this case might be classified under von Bechterew's type; but the root symptoms were not marked until an acute exacerbation of the disease occurred; the disturbances of sensation, the atrophy, the loss of the knee-jerk, may be interpreted as symptoms due to increased irritation or compression of the spinal roots. The enormously thickened membrane would give some hint as to the character of the morbid process, but we shall attempt no further inference from this, except to put the query whether the removal of the laminæ and of a piece of the thickened membrane, with the consequent release of pressure, may not have been responsible for the slight improvement in the patient's condition.

*Case 4.* Was seen in the clinic of Dr. Sachs. The patient is 51 years of age; a depot-master; born in England. The family history is entirely negative. He was always well, with the exception of a luetic infection 30 years ago, followed by secondary symptoms. A number of years ago had inflammatory rheumatism, lasting for over 3 months. His present complaint began about 3 years ago with pain across the small of the back. It was intensified by every movement of the leg or of the foot, but when pressure was applied over the painful parts or over the sciatic nerves, he did not experience any increase of pain. By degrees he began to walk stiffly, and he noticed an utter impossibility to flex the thigh upon the trunk, or to bend the spine. In rising or in sitting down had to exercise special precautions. At the time of our first examination, it was found that there was no pain over the sciatic, but the right iliac region was painful on pressure. The right leg is smaller than the left, but this is due, as the patient states, to an old fracture. There was an edematous swelling around the ankle, which was explained sufficiently by the report of the examination of the urine, which stated that there was considerable quantity of albumin and sugar. The pupils reacted slowly to light; all other movements were normal. The knee-jerks were increased, and ankle-clonus was present on both sides. Ever since the trouble began the patient has had some difficulty in micturition, and of late had some retention, dribbling from overflow.

During an examination made at the clinic the rigidity of

the spine was most marked, so that the patient could not lie on his face without having excessive pain.

In view of the previous rheumatic and specific history, it was considered advisable to place the patient on a thorough course of the iodides, and since last March, the time of first examination, a very marked improvement has taken place, so that there is to-day much less rigidity than there was last spring. The patient is now able to rise with greater ease, and is able to walk about with far less discomfort. The bladder symptoms have remained very much the same, and the condition of the reflexes is not improved.

In view of the preceding symptoms, the diagnosis of a specific or rheumatic pachymeningitis was the most plausible one, and the case is cited here simply to prove how closely this patient's condition resembles that described by von Bechterew, with the exception that the lumbar region and the hip joint are affected rather than the cervical region and the shoulder joint.

It would surely be an easy matter to increase the number of these histories, for we have all seen cases of chronic rigidity of the spine. It is fair to ask whether the authors who have raised this discussion have described anything distinctly new. Von Bechterew's type must for the present, and on the strength of his own statements, be regarded as a secondary form of spinal rigidity; and if we once begin to describe secondary forms, we shall have to include rigidity of the spine due to syphilis, to injuries, to rheumatism, to arthritis deformans, or in association with a number of chronic spinal cord affections. It is of great interest to know that a marked deformity of the vertebræ may result from a spinal cord affection, but there does not appear to be sufficient reason, for the present, to regard this type of von Bechterew as a distinct clinical entity.

But what are we to say of the Struempell-Marie type? There is an apparent resemblance and certain striking differences between this type and the ordinary rheumatic affections. First of all, let us note that in the majority of cases of articular rheumatism, or of arthritis deformans, the vertebral joints escape and the smaller joints are the ones most commonly affected. In articular rheumatism there is distinct hereditary predisposition, which has not yet been established with regard to the type in question. Rheumatic affections are characterized by frequent remissions, whereas "spondylose rhyzomé-

lique" seems to be practically a progressive disease. Ordinary rheumatic affections occur about equally in men and women, whereas the other disease has until now been noted chiefly in men. It might also be stated that the one is amenable to treatment, and the more recently described affection does not show a tendency to improvement, although Hoffmann has recorded such in his case. As for the resemblance to arthritis deformans, it need only be said that though the latter affects the smaller joints chiefly, it is also progressive.

A comparison of the pathological conditions of the joints in rheumatism and in the form of spondylitis in which we are interested, brings out the fact that in one the synovial membrane is the chief seat of the disease, the affection of the ligaments being secondary; but there is no such proliferation and ossification of the joint as has been noted in "spondylose rhyzomélique." On the other hand, if we take into account the appearance of the joint in arthritis deformans, we must concede that the differences are not so striking. We get hypertrophy of the ligaments in both, the only difference being in the marked ossification of the joint in the one form, while ankylosis occurs in arthritis deformans, as well as in the Struempell-Marie type.

The morbid changes causing "progressive ankylotic rigidity of the spine" appear to differ in degree and in localization, not in kind, from those found in extreme forms of articular rheumatism and arthritis deformans; from the clinical standpoint, however, and as an incentive to a further and more thorough study of rigid spines, "spondylose rhyzomélique" merits careful attention.

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EIN FALL VON ANGEBORENEM MYXEDEM. Besserung durch Behandlung mit Schilddrüsen-tabletten (A Case of Congenital Myxedema. Improvement Through Treatment with Thyroid Extract). F. Sklarek (Berl. klin. Woch., 16, 1899).

A case of cretinism is presented in this paper. The patient, aged 16, had been placed in a school for feeble-minded children, but showed no improvement. Under thyroid extract both physical and mental improvement were very decided. In hopes of establishing menstruation oophorin was administered. Two hundred tablets (.05) were given in four weeks without result. The thyroid extract was omitted for a time, but the development of the myxedematous condition was so marked that the treatment was resumed.

McCarthy.

MERALGIA PARESTHETICA (ROTH), WITH THE REPORT  
OF TEN CASES.<sup>1</sup>

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In 1895, Bernhardt, in a paper published in the *Neurologisches Centralblatt*, recorded a number of cases presenting a curious symptom to which at that time attention had not hitherto been directed. This consisted of a disturbance of sensation in a small oval area on the outer side of the thigh. It usually affected one leg, was characterized by burning, tingling, numbness or distinct subjective pain, and the presence of more or less disturbance of sensation—usually a diminution of one or more of the forms. Bernhardt had noticed this condition some years earlier and had reported a case that occurred in a young man suffering from bilateral ulnar neuritis, and several cases that had developed during convalescence from typhoid fever, but he had not at that time recognized the condition as an independent disease. One of our own cases and several published by other writers, Pieraccini, and others, were observed and noted before Bernhardt's first publication on this subject, but like his first case were not described as distinct morbid entities. A few months later, Roth published, in a separate monograph which he had previously read before a Russian medical society, the records of 15 cases. He named the condition meralgia paresthetica, and gave an analysis of the symptomatology to which little that is essential can be added. Since then, a number of cases has been reported by various writers.

We have been able to collect from the literature 89 cases. Sabrazes and Cabannes have collected 62, but as it is impossible to verify this number by the analysis of their paper, it is probable that some which were used in the compilation of their sta-

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<sup>1</sup> Read before the Philadelphia Neurological Society.

tistics were not described. It is not unlikely that a number of other cases have escaped our observation, for it is remarkable in what out-of-the-way journals they are usually reported. Freund, and Nücke agrees with him, believes that the disease is more common than this paucity of reports would indicate, and states that his colleagues, Rosenberg, Breuer, and Wandsbecker have, in conversation with him, cited cases that they had personally observed, but had not published; but Traugott argues that the number of cases reported is remarkably few, and a man with such an extensive experience as Osler has never seen a typical case. Meralgia paresthetica has attracted considerable attention among neurologists and even general clinicians, and the symptoms are frequently so intense as to disable the patient and oblige him to seek medical advice. It therefore seems probable to us that it is not a very common disease. Many continually on the watch for it never see a case, and the number recorded in the four years since Bernhardt's original publication is comparatively very small. It must be noted, moreover, that in the majority of papers, a single case only is described, and many of them are by men with large opportunities for clinical observation. As these men are familiar with the condition, one would expect them to have collected more cases and to have put them on record. We desire to add the records of the following cases to those already published.

*Case I.* R. A. M., male, 28, was admitted to the Presbyterian Hospital, October 30, 1898. The patient had been in the volunteer service of the United States, and had remained in one of the camps on American soil. Upon admission, the symptoms of typhoid fever were present; the Widal reaction was positive; the diazo reaction negative. In twelve days the temperature became normal. Three weeks later, he had severe vertigo, nausea and vomiting; he rapidly became intensely jaundiced, and there was pain in the right hypogastrium with diminution of liver dullness. Ten days after the initial symptoms, he had a severe chill, and developed facial erysipelas. Eleven days later, when the convalescence appeared to be established, he complained of tingling and numbness of the outer sides of both thighs. This became more and more severe, causing considerable discomfort and obliging him to keep quiet. Examination, a few days later, revealed an area of anesthesia on the right thigh extending on a line drawn from the external trochanter to the head of the fibula, from the ninth

to the thirty-seventh centimeter mark. The area was about 2 cm. broad above, 10 cm. broad in the middle, and 5 cm. broad below. Tactile, pain, temperature, and hair sensations were very markedly diminished in this area, but not abolished. These changes extended, gradually diminishing, for about 2 cm., into the surrounding skin. The sense of localization was imperfect, the error sometimes amounting to 5 cm. Localization was also impaired, but to a less degree, in the rest of the leg. Just below the anterior superior spine of the ilium, pressure caused sharp pain. On the left thigh, the area extended from the 6th to the 37th centimeter mark, and faded to nothing below. The disturbances of sensation were much the same, but the patient declared that they were subjectively less. Pressure just beneath the left anterior superior spine of the ilium caused severe pain. The patient had been obliged to wear a heavy belt, which possibly had sunk down and pressed upon the external cutaneous nerves. Absolutely no subjective sensation had been caused by this, and it was not until the 77th day of his sickness, counting from the time that he was unfit for duty in the military camp, that any pain was noted in the region of the thigh. The subjective sensations consisted of pain and numbness, more pronounced after exertion, and therefore towards evening. After the application of faradic electricity, the subjective sensations increased for a brief interval, and then became very much less. This treatment was regularly instituted, and caused rapid improvement; the duration of the symptoms being about four weeks. There was no neuropathic nor gouty trace in his family history.

*Case II.* J. K., male, 30, a clerk, is a man of regular habits, who has smoked to excess in early life, but denies alcoholism and venereal disease or excess. He is accustomed to considerable exercise, chiefly indoors. In 1895, he had a severe attack of diphtheria. About one year ago, he began to suffer with a curious sensation on the outer side of the right thigh. An area was found extending from the 8th to the 36th centimeter mark; 3 cm. broad above and of equal width throughout. In this area, touch, pain, temperature, and localization sensations were greatly diminished. In the corresponding area of the left thigh they appeared to be normal. The faradic current is not felt in this region, even when so strong as to be unbearable in any other part of the body. Curiously enough, sensation to the faradic current is also blunted on the left thigh, and there is a small area, corresponding to the central part of the area on the right side, where it is not felt. Pressure beneath the anterior superior spine of the ilium is not painful. Surrounding the anesthetic area is a zone in which the sensory disturbances are less pronounced. The subjective sensations are numbness, tingling, and pricking. They are not severe, but appear to be

rendered worse by wet weather, and after exercise. There is no history of injury, the symptoms appearing to have developed gradually. The patient is somewhat hypochondriacal; his father was eccentric, and indulged to excess in alcohol during the later years of his life. He had a habit spasm of the hand. During his adult life he suffered from time to time with paroxysms of numbness and tingling in the outer sides of the thighs, that would be so severe that he was disabled for several days at a time. These paroxysms were usually associated with exacerbation of gout.

*Case III.* J. A., a distinguished jurist, 76 years of age, a man of unusual intellectual ability. He was emotional, but did not use alcohol nor tobacco to excess. His appetite was large and he indulged it freely. He suffered with dyspepsia, and had repeated attacks of gout and eczema. About his 60th year, he began to suffer from pain and paresthesia on the outer aspect of the left thigh in the region of the distribution of the anterior crural nerve. At times, the hyperesthesia was so great that the mere contact of his clothing became unendurable, and he was obliged to stop walking in order to pull his garments from the skin. These paroxysms occurred irregularly, and were always very severe during stormy weather or after overwork. When examined in his 70th year, Heberden's nodes were present on the fingers; there was a systolic mitral murmur, and slight dilatation of the heart. Subsequently, he became senile, had attacks of despondency, and was quite hypochondriacal. *This patient was the maternal grandfather of the patient in Case II.*

*Case IV.* Miss L., 41, has had severe neuralgia for some years. She has also had neurasthenia, frequent attacks of gout, and dysmenorrhea. Her father, mother, and brother were also gouty. At her menstrual periods, she experiences an uncomfortable sensation on the outer aspect of both thighs. This consists of burning or numbness, and is associated with tenderness that persists during the intervals; the latter symptom is often quite severe. The paresthesias disappear with menstruation.

*Case V.* F. L., 24, complained in the second month of pregnancy of pain, numbness, tingling, and burning on the outer aspect of the left thigh. Her previous history is negative. She has suffered from various paresthesias in the hands and feet. Her father is living and in good health. Her mother suffers from melancholia, and is extremely gouty and neurotic. The symptoms continued until the termination of pregnancy, becoming so severe after the seventh month, that she was obliged to remain in bed. After delivery, the paresthesias became less severe, but are still present. The skin is not sensitive to touch, but pressure causes considerable pain.

*Case VI.* Miss L., 22, has had three attacks of acute vesicular eczema. On the fourth day of the third attack, she experienced a feeling of numbness, localized at a point 10 cm. above the head of the fibula on the outer side of the left thigh, that was relieved by a curious bursting sensation. This succession of symptoms occurred three times, and left a persistent feeling of numbness in an area 7 cm. by 11 cm. on the outer side of the left thigh, in which there is marked diminution of pain and tactile sensation. The left external cutaneous nerve is tender. The patient has had rheumatism, and is of neurotic temperament. She relates that for many years her father had persistent tenderness on the outer side of the left thigh, with paroxysms of pain that caused him sometimes to cry out as if struck. He died of tuberculosis. The mother has had neurasthenia in a severe form.

*Case VII.* Mrs. F. H., aged 23, had excruciating pains in the middle of the thighs, particularly on the outer aspect, which developed two hours before the occurrence of the signs of parturition. They disappeared after delivery, but recurred in a milder form at every menstrual period, and during a second pregnancy were constant throughout. In the involved areas, touch, temperature, muscle, and localization sensations were all good.

*Case VIII.* M. M., male, 50, has been obliged to do a great deal of walking during the past three or four years. He has noticed a peculiar sensation of coldness on the outer side of the right thigh in an area occupying the middle third. Occasionally, there are subjective sensations of tingling and numbness. The pains are worse at night and in cold weather; and exposure to cold invariably produces a severe attack. In the affected area, there is complete anesthesia and analgesia. A similar condition is also found in the left leg restricted to a smaller area. The nerve trunks are not tender. The patient complains, however, of occasional shooting pains in the legs, both knee-jerks are exaggerated, the Achilles tendon reflex is present, station is normal, and the pupils react sluggishly to light. In addition to the excessive walking, the patient had had, a short time previously, a severe attack of pneumonia. Later, after the disease was partially developed, he had a severe injury to the left leg followed by another attack of pneumonia. Two attacks of sciatica occurred after the appearance of the paresthetic symptoms. There is apparently no neuropathic family history.

*Case IX.* A distinguished physician of this city has suffered from a feeling of numbness and burning in the posterior aspect of the right thigh for years. At times he also has a sensation as if some wet object were in contact with the skin.



The symptoms followed a severe attack of influenza that occurred seven years ago.

*Case X.* M. F., male, white, about 60 years of age. The patient is an apparently healthy man, without neuropathic heredity, and without gouty tendencies. He has never indulged in stimulants to excess, and denies all constitutional diseases. His father died of old age; his mother had a stroke of apoplexy about the age of 50. Thirteen years ago, shortly after a severe attack of typhoid, he noticed from time to time attacks of peculiar sensation in the left thigh, characterized by a feeling of pricking with slight numbness. This occurred on the outer side of the thigh, and on the anterior surface just above the knee. The pain was never very severe, he was never obliged to rest on account of it, and it rarely lasted more than one-half a day. It was not increased by exercise or standing, and apparently not relieved by rest. Hot applications caused temporary amelioration. The attacks are usually more frequent in winter and somewhat more severe. As far as known, they are not brought on by excessive exertion, by exposure to cold, by heating, or by slight attacks of infectious disease. At present, there are no objective disturbances, there is no tenderness upon pressure beneath the anterior superior spine of the ilium, there is no disturbance of tactile or localization sense in the thigh, but there are two distinct areas of hypalgesia; one 13 cm. by 5 cm. on the outer surface in about the middle—the other 9 cm. by 1 cm. in the median line just above the knee. These areas fade gradually into the surrounding skin where the sensation is normal.

Meralgia paresthetica is a disease that appears to be peculiar in its clinical manifestations from any of the ordinary forms of neuritis or neuralgia. The description of paresthesia of the region supplied by the external cutaneous nerve of the thigh is fairly accurate, but it omits any reference to another common and peculiar feature of the case, that is—the objective disturbance of sensibility. Perhaps as good a definition as any that could be given would be to call it *a disturbance of sensation on the external surface of the thigh, characterized by various forms of paresthesia, associated with dissociation and more or less diminution of sensation.* The earlier writers called attention to the fact that men were more frequently affected than women, and Stembo has recently again brought this fact into prominence. Our own statistics fully bear out this view. Altogether, including our own, we have been able to collect 99 cases, of which 75 were in men, 21 in women, and in 3 the sex was not

given. In our own cases, however, this proportion was not maintained, 6 only being men, and 4 women. Two factors probably contribute to cause this marked disparity; first, the greater exposure to which the male sex is liable, the number of cases developing after traumatism being very considerable; second, the fact that women are far less likely to call attention to a symptom of this nature, partly on account of modesty, and partly because their occupations, being more sedentary, do not make the disease so disabling as it is in the other sex. The distribution of the symptoms is less arbitrary. In the great majority they occupy the region of distribution of the nerve. In several of Roth's cases, in Lop's case and in one of ours (Case X) there were disturbances in the anterior as well as the external surfaces of the thigh. In Escat's case, the inner sides of both thighs were also involved. In one reported by Nartowski, there were paresthesia in the leg and feet, as well as in the thigh, and Osler has reported three cases characterized by peculiar paresthetic symptoms in one or both legs which, however, did not present alterations in the outer side of the thigh. One of these cases was remarkable in that in some respects it resembled akinesia algera. It has been noted by several observers that when one side is involved, the corresponding region upon the other side shows more or less objective disturbance of sensation, particularly to the faradic current. In the majority of cases, unfortunately, it is probable that this symptom was not investigated. In one of our cases investigation of this point led to the discovery that the disease was really bilateral, that is to say, there were slight objective disturbances also upon the other side. Köster has also reported a similar case. In very few of the cases described as bilateral were the symptoms equally severe on both sides, and there appeared to be no difference as to which was more severely involved.

Why the external cutaneous femoral nerve should be the only one in which this peculiar syndrome occurs, has excited considerable speculation. Breuer was the first to suggest, in a verbal communication to Freund, that the anatomical relations of the nerve to the surrounding structures might have something to do with this, particularly on account of the superficial situation of the nerve, and its exposure, therefore, to in-

jury. Since then, the majority of writers upon this subject have adopted this view and some have added to it that perhaps not only was the nerve exposed to traumatism, but the fascia, under certain circumstances, might press upon it and cause the anesthesia and the irritation. This would be particularly likely to occur if the nerve occupied a slightly anomalous position; as in Hegar's case, where it lay about 3 cm. from its usual situation. It does not seem to us, however, that this argument is entirely satisfactory. It is true that the external cutaneous femoral nerve occupies an exposed position, but so do many other nerves of the body, and nerves that are on the whole more liable to injury. Yet this syndrome practically is not observed anywhere else, excepting perhaps in the anterior crural nerve; the anterior form of meralgia paresthetica according to Roth; but very few cases of this condition are reported. The most typical cases are those of Féré and Möller. In the two cases in which histological examination of the nerve has been possible, evidences of change were present only in one, and pressure sufficient to cause the extreme symptoms in the other case (that of Souques) would almost certainly have produced degenerative changes in the nerve fibers. If this nerve were so particularly exposed, there is no reason why meralgia paresthetica should not be considerably more common than appears to be the case. It must be remembered moreover, that it has never been demonstrated that the fascia does or could press upon the nerve trunk; and it is therefore a pure assumption to regard this condition as a cause.

The etiology is indeed so various as to render us rather skeptical regarding the real causative factor of some of the agencies that have been mentioned. Among the predisposing causes, Stembo mentions a sedentary life. Shaw and others call attention to the frequency with which constipation occurs. It is mentioned of many of the patients that they were obese, that a rheumatic diathesis existed, or that they were addicted to alcohol; but in others none of these conditions existed. Bernhardt, in his original publication, called attention to the frequency with which various infectious diseases precede the development of the symptoms, the most common being, apparently, typhoid fever.

A case of the latter disease under our care, developed in the

course of convalescence a curious symptom that may have some bearing upon this question. The patient, a girl of twenty, had had the disease in very severe form, with an almost equally severe relapse. Throughout the whole course of the case, the nervous symptoms had been very prominent. There was much delirium, exaggeration of the reflexes, flexion of the limbs, and pain when an attempt was made to extend them. During convalescence, she developed tender toes. The fever had disappeared, but while the patient was still very weak, she complained one day of paresthetic sensations on the outer side of the left thigh. Examination showed diminution of sensation in the usual area and exquisite tenderness over the trunk of the external cutaneous nerve, just below the anterior superior spine of the ilium. The next day, all the symptoms had disappeared, with the exception of the tenderness over the nerve trunk, which persisted for several days longer. It appears as if in this case there had been a transient peripheral neuritis of the nerve trunk of very mild form.

Among the other infectious diseases, syphilis has been noted as occurring as an antecedent condition in many cases, and Bacelli has reported two cases and Bernhardt and Pitres each one, in which locomotor ataxia was present; and Möbius has mentioned two cases with beginning general paresis. In one of Stembo's cases, traumatism seemed to play a prominent part. In several reported by Bernhardt and one by Devic, continuous irritation produced by the striking of the sword against the thigh, was held to be responsible. V. Lutzenberger believes that an old injury may predispose the nerve to inflammatory processes, and constitute it a *locus minoris resistentiae*, and that when this has occurred, an infectious process is likely to set up a neuritis. In his own case, this appeared to have been so, and in one of ours, there was much reason to believe that a heavy army belt had pressed down upon the anterior superior spines of the ilia, and wounded the nerve, and that later a succession of infectious processes was responsible for the development of the condition. Dopter has reported cases associated with other pathological conditions producing injury to the nerve, as Pott's disease, tumors, etc. Allied to traumatism is pregnancy, which is certainly responsible for more cases than have hitherto been recorded.

The importance of a neurotic predisposition has not been sufficiently investigated, but it appears that many of the cases have this characteristic. It is, of course, widely distributed, and it is difficult to say whether a greater proportion of cases with neuropathic heredity suffer from meralgia paresthetica than would occur in a number of persons taken at haphazard or not. It is certain that in women, the symptoms are usually worse during menstruation when the nervous system is distinctly more irritable. Until our own, no cases were reported in which a distinct hereditary influence could be traced. In Case II. the father of the patient had suffered for a number of years from exactly the same condition, and the maternal grandfather had also been a sufferer from the disease in his old age. This is a remarkable example of similar cumulative ancestral influence. The patient developed the symptoms much earlier than either his grandfather or his father had done. In Case VI. the father of the patient had also suffered from meralgia paresthetica for a number of years.

Finally, a number of cases have followed the application of hydrotherapy, cold douches to the back or legs being particularly likely to provoke the disease. This fact has been used to explain its occurrence after typhoid fever, but it does not appear that it is more frequent now than formerly, and the use of cold water in the treatment of this disease is much more prevalent than even in 1895. Venturi alone of all the writers on the subject has suggested the possibility of a cerebral origin of the disease, locating the lesion in the thermo-perceptive centers. In his two cases the alterations in the temperature sense were very pronounced.

The pathology is equally doubtful. One case has come to autopsy, and this is so important that it deserves a full description. The patient, 80 years of age, who was observed by Nawratski, was an inmate in the lunatic asylum. It is recorded that he had an objective diminution of touch and temperature sensation, with preservation of the pain sense in the affected area. At the autopsy, a spindle-form swelling was found in each nerve at the point where it crossed the crests of the ilia. In the region of these swellings, there were the changes characteristic of neuritis and perineuritis, and peculiar lesions that Nawratski considers to be the result of an old chronic inter-

stitial neuritis. Secondary degeneration of the nerve fibers extended in both directions, but the degenerated fibers were more numerous in the peripheral than in the proximal portion of the nerve. The spinal cord was examined, and found to show slight sclerosis in the posterior columns, particularly on the left side. As a result of this examination, Nawratski accepts the mechanical theory, and believes that unilateral, or even bilateral involvement of the nerve occurs, which is usually the result of some old injury whose occurrence may perhaps be favored by the anomalous course of the nerve. In one other case, that of Warda, it was noted that the nerve trunk on the affected side was swollen and tender, and distinctly palpable. In none of our cases could the nerve be felt, and no other author has recorded a similar peculiarity.

Bernhardt, in his last publication on the subject, notes that in the majority of cases, there is tenderness on pressure just in front of and below the anterior superior spine of the ilium. Unfortunately, this symptom has been tested in only a few cases. Nartowski found it in one case out of five, Devic in his case, but Köster, Igelrans, and Pitres distinctly state that the nerve trunks were not tender. In two of our cases, in which they could be tested, a similar condition was present. In the case of typhoid fever with transient symptoms, they were distinctly tender, and remained so for some time, and in three no tenderness existed. Devic, to whose paper we believe sufficient attention has not hitherto been paid, was the first to suggest the occurrence of this symptom. He goes on to argue, however, that the absence of trophic changes is against the existence of a neuritis; and, in fact, eliminates destructive lesions of the nerve itself. He is also disinclined to regard the condition as neuralgic, on account of the absence of the typical neuralgic symptoms and the difference in the general character of the pain, and the very pronounced objective disturbances of sensation that are present. He suggests, therefore, the possibility of a periostitis of the femur, a suggestion that has not, and we believe correctly, received any favorable consideration. At any rate, in the absence of objective alterations in the nerve itself in the great majority of cases, and until a number of autopsies prove the contrary, we feel that Devic is justified in believing that a neuritis is not invariably present.

Extra-neural pathological conditions have occasionally been responsible for the condition. In one of Doppter's cases, a tumor pressed upon the nerve trunk. In another, a deformity of the spinal column, resulting from caries, compressed it. In both cases, partial degeneration of the nerve was found. In Souques' case, the resected portion of the nerve was examined carefully and found to be entirely normal.

Aside from the suggestions of Venturi and Devic, no writer has supposed that the symptoms were due to anything except a lesion of the external cutaneous nerve itself. Lop, however, mentions that in his case, the skin of the lower third of the thigh was red, purple, warm, rough, and that it could not be pinched into folds; there does not appear to have been edema of the foot. The sensory disturbances occupied the outer side and lower anterior third of the thigh, the same distribution found in Case X of our series, whose skin was perfectly normal. In Rapin's case, the skin in the analgesic region was abnormal to the extent of not assuming the goose-flesh appearance when cold was applied; the surrounding skin was normal. This is rather an evidence of nervous disturbance than of any lesion of the skin itself.

The symptoms of the disease are so variable that it is difficult to describe them. In the majority of cases during the early stages, there is tingling, increasing to positive pain, usually commencing when the patient is standing or walking, and continuing until he or she sits down and flexes the thigh upon the abdomen. In other cases, the patients complain of a sensation of numbness, or of cold, or of aching, or they may feel as if a wet cloth has been applied to the skin, and sometimes there is tenderness so exquisite that even the pressure of the clothing can not be borne, as in cases reported by Köster, Osler, and ourselves. The symptoms in the great majority of cases first appear after the patient has had the fascia lata tense for some time, and this leads to the theory, that the condition was due to pressure upon the nerve by this fascia; but in several cases the symptoms persisted when the patient was lying down, and sometimes were most severe at night. The objective disturbances are usually unequal diminution of various forms of sensation. The most profoundly affected are usually pain and electro-cutaneous sensation, then tactile sensation.

thermic sensation, pressure and localization. In the early stages, however, there may be hyperalgesia, and often exquisite tenderness persists throughout the whole course of the disease. V. Lutzenberger, as a result of the analysis of his own case and that of Freund, reaches the conclusion that, in early stages, there is hyperalgesia with anesthesia to cold; in the late stages analgesia with anesthesia to heat; but this theory is not borne out by the examination of the other cases. Usually around the area most intensely affected, there is a zone through which the objective disturbances gradually diminish. The sciatic nerve is never tender. Sabrazes and Cabannes note that in sciatica, pain is increased by flexion of the thigh upon the abdomen, particularly if the leg is extended at the knee-joint, and that in *meralgia*, the reverse is the case, and in *neuralgia* of the anterior crural nerve, the nerve itself is often tender, and the pain spreads over the anterior and inner surface of the thigh.

Akinesia *algebra*, or the allied condition *apraxia algebra*, could only in rare instances cause any difficulty. In either, the absence of objective sensory disturbances, the irregular localization of the pain or paresthesia, and the presence of hysterical or degenerative stigmata should be sufficient to determine the true nature of the case.

The differential diagnosis from intermittent claudication, as described by Charcot, Goldflam, Bieganski, and others, is still more difficult. According to Charcot, in this condition, the patients, although perfectly comfortable when lying down, when they attempt to walk, develop a feeling of weight and discomfort in the leg which becomes slightly cyanosed and at times so painful that further locomotion is impossible. Charcot associated this condition with diabetes and arterial obstruction. Goldflam, who reported a number of cases, noted that it occurred usually late in life, and that when the attempt to walk was made, there rapidly developed a feeling of fatigue and then pain with paresthesia, chiefly in the leg and to a less extent in the thigh. The patients were nearly all cases of marked arterial sclerosis, and in many cases, the reflexes were exaggerated, and there was ankle-clonus. The distribution of the symptoms and the age at which they develop are the most important distinguishing characteristics.



The course of the disease is extremely variable. In some of the cases the symptoms persist for years, but exhibit considerable variation in intensity, in others they disappear after a few months. In a number of instances, recurrent attacks have been mentioned. As the majority of cases have not been observed for a sufficiently long period to enable us to control the results of treatment, very few cases of recovery have been recorded. Näcke, Köster and Souques have each reported one and we have reported another in which this occurred. In our case, however, the patient passed from observation about a month after the symptoms had disappeared, and it is impossible to be certain that the recovery was permanent. None of our other cases have shown any improvement, although in those brought on apparently by pregnancy, the symptoms diminished after delivery. Roth, Feund, and a number of others have secured more or less permanent improvement by treatment. Good reports a case with a typical relapse six years after the cessation of the previous attack, and Köster reports another in which the period was eight years. In the majority of cases, the symptoms are not persistent, but disappear during rest and increase during standing or exercise; but in some cases, the pain diminishes during walking. Nartowski reports a remarkable case in which the pain increased during sleep. In Ingelran's case, the symptoms appear to have been paroxysmal, but no cause could be assigned for their development, for the attacks were not altered either by rest or action. In many of the cases, exposure to cold, as in one reported by us, seems to have been of more importance than the mere act of walking. The prognosis must, therefore, be exceedingly grave for recovery or distinct amelioration. Of course the disease itself is of such trivial nature that even injurious action upon the general health of the patient could rarely be produced.

The treatment, in view of the almost uniformly bad results, is anything but satisfactory. When the disease was considered to be rheumatic in nature, salicylic acid was employed rather freely internally, and Devic used inunctions of this drug. Counter-irritation over the area almost invariably produced bad results, and was soon abandoned. Roth recommended, however, the application of faradic electricity, particularly in the form of the dry brush, and secured very excellent results by

this means. In the case of ours which recovered, we employed this form of treatment exclusively, and after each application, the patient declared that he felt distinctly benefited. Massage has also been extensively used, but the reports of its employment are somewhat contradictory. When the symptoms are exceedingly severe, it is possible that operative interference would be justifiable, although in the absence of an accurate knowledge of the pathology of the condition, these measures should be delayed as long as possible. Spiller has declared himself in favor of resection when other means have failed, or, if milder measures are thought desirable, simply stretching of the nerve trunk.

Souques has recently reported a case in which this operation was performed, apparently with entire success. The patient, a girl of 21, had been obliged to stand and work hard for a number of years. On one occasion, after unusual fatigue, she noticed tingling and chilliness on the outer side of the left thigh, which was relieved by sitting or lying down. Fourteen days later the pain became very severe and finally required hypodermic injections of morphia. It was observed that the puncture of the needle was not painful and investigation then showed that there was complete anesthesia to all forms of sensation in an area extending from the trochanter to the knee. Later the paroxysms of pain became so severe that the patient was unable to work, and all other forms of treatment proving unavailing, several centimeters of the anterior crural nerve were resected. There was complete relief, but the disturbance of sensation persisted in the area of the distribution of the nerve. Histologically, the nerve trunk was normal.

An interesting case, that is probably of this nature, in which this treatment was employed, was reported by Hager as far back as 1885. A girl of 21 received a severe injury to the left hip while dancing. From that time she had continuous dull pain in the left hip and thigh, was able to walk very little, and suffered greatly in general health. There was marked tremor of the left leg when she attempted to lift it from the bed. Examination showed no apparent lesion, there was extreme hyperesthesia over the external surface of the left leg and a tender point 3 cm., outside the anterior superior spine of the ilium. An incision was made at this point, and an anomalous

external cutaneous femoral nerve was found and resected. The patient was relieved of her subjective sensations, but there was complete anesthesia in the area of distribution of the nerve.

In all likelihood, the most important feature of any form of treatment would be the insistence upon absolute rest in bed for a considerable period of time, but few of the patients can be persuaded to submit to this.

If the attempt is made to classify meralgia paresthetica, it is found that there is considerable difficulty in including it under either the neuralgias or the neuritides. From the former, it differs by the fact that pain is usually a subordinate symptom and often not present; whereas, the various forms of paresthesia are of paramount importance. Moreover, it is exceedingly rare in any form of neuralgia to have the pronounced objective disturbances of sensation that are almost invariably present. From neuritis, it differs by the fact that the symptoms are usually persistent, and rarely progressive or retrogressive. The nerve trunk, aside from the tender point just beneath the spine of the ilium that is occasionally, but not frequently, present as Bernhardt contends, is not tender. Trophic changes never occur, and there is often pronounced dissociation of sensation, which is at least rare in neuritis. In many respects, however, it can be regarded as resembling the occupation neuroses, excepting that these are not limited to the distribution of any particular nerve. The abnormal sensations are usually brought on by effort and disappear upon rest. The condition is practically incurable, is often associated with overwork, as prolonged standing or walking, with the neuropathic diathesis, or it is occasionally hereditary. A patient of one of us, a young woman of 20, with severe writer's cramp, is the daughter of a man who has suffered for years from the same condition. Its similarity and differences from the intermittent claudication of Charcot and Goldflam have already been mentioned. If it were permissible to speak of a neurosis of a nerve, we believe that that would be the most fitting designation. At present, however, it is only possible to say, that it is a distinct morbid entity from the clinical standpoint, but that a variety of causes may, in different cases, be concerned in its production.

## CASES OF MERALGIA PARESTHETICA.

| Sex.                  | Age. | Side.   | Cause.   | Duration.   | Remarks.  |
|-----------------------|------|---------|--|-------------|---|
| <i>Bernhardt.</i>     |      |         |  |             |   |
| 1                     | m.   | 51 L.   | Typhoid, striking of sword against leg.              | ?           | Army officer.   |
| 2                     | m.   | ? L.    | Striking of sword against leg.                       | ?           | Army officer.   |
| 3                     | m.   | 56 L.   | Cold douches.  | 4 yrs.      | Tactile sense diminished.   |
| 4                     | m.   | 30 L.   | Chronic lead poisoning.                              | ?           | Skin and underlying muscles tender, numbness.                     |
| 5                     | m.   | 35 R.   | None. Pedler; exposed to oppressive heat.            |             | Numbness.   |
| 6                     | m.   | 34 R.   | None. Blacksmith.                                    | Few weeks.  | Numbness.   |
| 7                     | f.   | 27 R.   | Pregnancy.   | ?           | No objective disturbances.  |
| 8                     | m.   | 50 L.   | Syphilis, followed by locomotor ataxia.              | ?           | Slight hypesthesia.   |
| <i>Wladimir Roth.</i> |      |         |  |             |   |
| 9                     | m.   | 41 L.   | Rough walking, cold.                                 | 8           | All forms of sensation diminished.                                |
| 10                    | m.   | 46 Bil. | Exposure to cold. Syphilis. Typhoid fever.           | 12          | Tactile hypesthesia. Improved.                                    |
| 11                    | m.   | 44 Bil. | None.  | 2           | Subjective hypesthesia only.                                      |
| 12                    | m.   | 55 Bil. | Influenza.   | ?           | Subjective hypesthesia.   |
| 13                    | m.   | 41 ?    | None.  | 10          | Paresthesia only.   |
| 14                    | m.   | 35 Bil. | Alcoholism.  | 4           | Slight hyperesthesia. The affected area remains pale in hot bath. |
| 15                    | m.   | 54 Bil. | Alcoholism, syphilis, infectious fevers, traumatism. | 4           |   |
| 16                    | m.   | 48 R.   | Typhoid fever.                                       | 6           | Slight manifestations.  |
| 17                    | m.   | 36 ?    | Exposure to cold.                                    | 12          | Hypesthesia to all sensations.                                    |
| 18                    | f.   | 28 Bil. | Nervous, anemic.                                     | 3           | Subjective hypesthesia.   |
| 19                    | f.   | 41 R.   | Eclampsia, epilepsy.                                 | ?           | Previous attack years ago.  |
| 20                    | m.   | 43 R.   | Syphilis, alcoholism.                                | 8           | No objective changes.   |
| 21                    | m.   | 45 L.   | Exposure to cold.                                    | 15          | Slight hypesthesia.   |
| 22                    | m.   | 35 R.   | Rheumatism, alcoholism.                              | 3           | Pains preceded by a sense of cold and pricking.                   |
| <i>Näcke.</i>         |      |         |  |             |   |
| 23                    | m.*  | ? R.    | Misstepped heavily on rt. foot. Pain later.          | 4 or 5 days | Recovery.   |
| <i>Freund.</i>        |      |         |  |             |   |
| 24                    | m.*  | 39 R.   | Cold douches.  | 7           | Improvement; symptoms extending downward.                         |
| 25                    | m.   | ? ?     | Syphilis.  | ?           | Not a case of locomotor ataxia.                                   |
| 26                    | m.   | 50 Bil. | Emotional disturbance.                               | ?           |   |
| 27                    | f.   | 58 ?    | No cause given.                                      |             |   |
| <i>Escat.</i>         |      |         |  |             |   |
| 28                    | m.*  | 31 Bil. | Long walking tour.                                   | 13 mo.      | Increased by standing or exercise.                                |

|                         | Sex. | Age. | Side. | Cause.   | Duration. | Remarks.  |
|-------------------------|------|------|-------|--|-----------|---|
| <i>Hirsch</i>           |      |      |       |  |           |   |
| 29                      | m.   | 50   | ?     | Alcoholic, syphilitic. Local injury, exposure.                                       | 2 mo.     | Burning pain. Diminution of the thermic sense.  |
| <i>Knauer.</i>          |      |      |       |  |           |   |
| 30                      | m.   | 36   | L.    | Striking of sword against thigh, fatigue, alcoholic.                                 | 12 yrs.   | Delayed sensory perception. Hypesthesia, hypalgnesia.   |
| 31                      | m.   | 43   | R.    | Alcoholic, long foot tours.  | ?         | Feeling as if flesh were loose. Subjective sensation of cold. Sometimes increased by drinking.  |
| 32                      | f.   | 22   | R.    | Childbirth, typhoid fever, followed by multiple neuritis and paralysis of both legs. | 4         | Hypesthesia, hypalgnesia.   |
| 33                      | m.   | 33   | R.    | Carried packages on the right side.  | ?         | Diminution of patellar reflex. No disturbance of sensation, excepting slight hypesthesia.   |
| <i>Stembo.</i>          |      |      |       |  |           |   |
| 34                      | f.   | 25   | L.    | Repeated pregnancies.  | 3 mo.     | Pain relieved by flexion of limb.   |
| 35                      | m.*  | 60   | L.    | Exposure in military service.  | ?         | No tenderness over nerve.   |
| 36                      | m.   | 25   | L.    | No cause given.  | 1 yr.     | Pain diminished by flexion. No tenderness over nerve.   |
| 37                      | m.   | 16   | R.    | No cause given.  | 2         | Pain diminished by rest or rapid walking.   |
| 38                      | m.*  | ?    | L.    | Severe injury to knee.   | 2         | General hypesthesia to all sensation including electricity. Slight hypesthesia in corresponding area of right leg.                    |
| <i>v. Lutzenberger.</i> |      |      |       |  |           |   |
| 39                      | m.*  | ?    | ?     | Injury 5 years before symptoms developed.  | 3 weeks.  | Tactile sensation normal; diminution of electrocutaneous sensation.   |
| <i>Köster.</i>          |      |      |       |  |           |   |
| 40                      | m.   | 64   | Bil.  | Long standing in museum.   | ?         | First attack in 1884. Relapse in 1892, when left leg was also involved. All forms of sensation diminished. No pain over nerve trunks. |
| <i>Adler.</i>           |      |      |       |  |           |   |
| 41                      | m.   | 49   | L.    | Long standing.   | 1         | Numbness.   |
| <i>Warda.</i>           |      |      |       |  |           |   |
| 42                      | m.   | 32   | L.    | Perityphlitis. Neurasthenia. Cold douches, bicycle riding.                           | 3 mo.     | Nerve trunk sensitive.  |
| <i>Shaw.</i>            |      |      |       |  |           |   |
| 43                      | m.   | 50   | R.    | ?  | 10        | Increased by standing. Period of almost complete remission. Slight symptoms on left side.   |
| 44                      | m.   | 50   | R.    | Syphilis, constipation.  | 3         | Involved area has gradually increased. Headache, vertigo, disturbance of gait, bradycardia.   |

|    | Sex. | Age.  | Side. | Cause.                                     | Duration. | Remarks.   |
|----|------|-------|-------|--|-----------|--|
| 45 | f.   | 36    | L.    | Childbirth, con-                           | ?         |  |
|    |      |       |       | stipation.                                 |           |  |
| 46 | f.   | 60    | L.    | No cause given.                            | ?         |  |
|    |      |       |       | <i>Good.</i>                               |           |  |
| 47 | f.   | young | L.    | Exposure to wet.                           | ?         | Collapse after 6 years, hyp-<br>esthesia, analgesia.   |
|    |      |       |       | <i>Traugott.</i>                           |           |  |
| 48 | m.   | 60    | R.    | Prolonged stand-                           | 20        | Hypesthesia of all forms of<br>sensation, including elec-<br>tro-cutaneous.  |
|    |      |       |       | ing.                                       |           |  |
| 49 | m.   | 55    | L.    | Gout, obesity.                             | 1         | Hypesthesia of all forms of<br>sensation, including elec-<br>tro-cutaneous.  |
|    |      |       |       | Prolonged walking.                         |           |  |
| 50 | f.   | 49    | L.    | ?  | ?         | Hyperalgesia. Sensation<br>otherwise normal. Sci-<br>atic not tender.  |
|    |      |       |       | <i>v. Nartowski.</i>                       |           |  |
| 51 | m.   | 51    | R.    | Rheumatism.                                | 26 yrs.   | Hypesthesia to all forms of<br>sensation, including elec-<br>tro-cutaneous. Nerve<br>trunk slightly tender.  |
| 52 | m.   | 45    | L.    | ?  | ?         | Hypesthesia of all forms of<br>sensation, including elec-<br>tro-cutaneous. Pain in-<br>creased by pressure, and<br>during sleep.                    |
| 53 | f.   | 63    | Bil.  | Doubtful.                                  | ?         | Hyperalgesia. Hypesthesia<br>to all forms of sensation,<br>including electro-cutane-<br>ous.   |
| 54 | m.   | 53    | Bil.  | Syphilis. Symp-                            | 15        | Symptoms extended to legs<br>and feet. Localization<br>and electro-cutaneous<br>sensation diminished.<br>Analgesia.                                  |
|    |      |       |       | toms followed an attack of pneu-<br>monia. |           |  |
| 55 | m.   | 48    | Bil.  | Cold baths, 8                              | 8         | Pain, pressure, and tactile<br>sense diminished. Elec-<br>trical irritability of the<br>muscles in this region<br>diminished. Skin reflexes<br>less. |
|    |      |       |       | douches.                                   |           |  |
|    |      |       |       | <i>Nawratski.</i>                          |           |  |
| 56 | m.   | 80    | Bil.  | Doubtful.                                  | ?         | Touch and temperature<br>sense diminished. Au-<br>topsy.   |
|    |      |       |       | <i>Spiller.</i>                            |           |  |
| 57 | m.   | ?     | R.    | ?  | 1         | Sensation normal.  |
|    |      |       |       | <i>Devic.</i>                              |           |  |
| 58 | m.   | ?     | ?     | Continual pres-                            | ?         | Sensation of moisture. Hy-<br>pesthesia. Tenderness of<br>the nerve trunks.  |
|    |      |       |       | sure against the thigh.                    |           |  |
|    |      |       |       | <i>Lucas-Champonniere.</i>                 |           |  |
| 59 | m.   | ?     | ?     | No cause.                                  | 8         | Symptoms have gradually<br>increased. There is a feel-<br>ing of congestion when<br>standing.  |

| Sex.               | Age.  | Side. | Cause.  | Duration. | Remarks.  |
|--------------------|-------|-------|---|-----------|---|
| <i>Bellot.</i>     |       |       |   |           |   |
| 60 f.              | 48    | L.    | Gout, varicose veins.   | ?         | Hypesthesia.  |
| <i>Donath.</i>     |       |       |   |           |   |
| 61 m.              | 40    | R.    | Injury and exposure to draught.   | 7         | Symptoms worse when walking or standing. All forms of sensation diminished. Distinct weakness of the right leg. No improvement.                       |
| <i>Pieraccini.</i> |       |       |   |           |   |
| 62 m.              | 50    | L.    | Vigorous. No neuropathic condition.   | ?         | Pain increasing with walking.   |
| 63 f.              | young | Bil.  | No neuropathic condition.   | ?         | Pain, severe enough to cause tears; brought on by walking.  |
| 64 f.              | 40    | R.    | Obese.  | 10        | A feeling of numbness.  |
| <i>Ingelrans.</i>  |       |       |   |           |   |
| 65 m.              | ?     | ?     | Gouty.  | ?         | Subjective sensation of cold, not altered by rest or action. Hyperalgesia. Diminished heat perception. Nerve trunks not tender.                       |
| <i>Dopter.</i>     |       |       |   |           |   |
| 66 m.              | 22    | ?     | Alcoholic, psoriasis, urticaria, long marches.                              | 12        | Pain disappears upon resting. Loss of sensation to cold. Unequal pupils. Electro-cutaneous sensation increased.                                       |
| 67 f.              | 40    | L.    | Varicose veins.   | 10        | Pain produced by walking; disappears upon rest. Reappears in bed. No disturbance of sensation, excepting slight hypesthesia during paroxysms of pain. |
| 68 m.              | ?     | ?     | Pott's disease at the 2d lumbar vertebra, causing compression of the nerve. | ?         | 1st symptom hypesthesia, followed by anesthesia without spontaneous pain.   |
| 69 m.              | ?     | Bil.  | Malignant tumor pressing upon nerve.  | ?         |   |
| <i>Verhoogen.</i>  |       |       |   |           |   |
| 70 m.              | 34    | ?     | Infantile paralysis.  | 4         | Pain occurs during walking, disappears upon resting.  |
| <i>Bacelli.</i>    |       |       |   |           |   |
| 71                 | ?     | ?     | A prolonged walk.   | ?         |   |
| 72                 | ?     | ?     | Symptoms preceded by locomotor ataxia.                                      | ?         |   |
| 73                 | ?     | ?     | Symptoms preceded by locomotor ataxia.                                      | ?         |   |
| <i>Pitres.</i>     |       |       |   |           |   |
| 74 m.              | 48    | Bil.  | Rheumatic. Influenza.   | 3         | Hypesthesia, diabetes.  |
| 75 m.              | 32    | L.    | Syphilis, light-nining pains.   | 12 days   | Anesthesia developed suddenly, hypesthesia and hypalgesia. Nerves not tender. Knee-jerks present.   |

|                   | Sex. | Age. | Side. | Cause.  | Duration. | Remarks.  |
|-------------------|------|------|-------|---|-----------|---|
| 76                | m.   | 45   | L.    | Obese, migraine, intestinal colic.  | 2         | Burning sensation brought on by walking. Nervous system otherwise intact.   |
| <i>Lop.</i>       |      |      |       |   |           |   |
| 77                | m.   | 30   | R.    | Typhoid fever. Bilious hematuria.   | 1         | Fulgurant pains. Walking only possible with aid. Areas of analgesia on external surface and lower anterior third. Hyperesthesia. Skin thickened and altered. Improvement. |
| <i>Souques.</i>   |      |      |       |   |           |   |
| 78                | f.   | 21   | L.    | Prolonged standing.   | 1         | Anesthesia to all forms of sensation. Resection of nerve. Cure.   |
| <i>Claisse.</i>   |      |      |       |   |           |   |
| 79                | m.   | 33   | R.    | Severe injury to knee.  | 5         | Paresthesia, area of anesthesia; retardation of sensation of cold. Knee jerks normal.   |
| 80                | m.   | 36   | R.    | Malarial infection.   | ?         | Pain severe enough to oblige patient to rest. Anesthesia on antero-external surface of thigh. Knee jerks normal.  |
| 81                | m.   | 61   | L.    | Soft chancres, no evidence of syphilis, paresis.  | ?         | Rapidly fatigued by walking. Area of anesthesia. Retardation of sensation of cold. Knee jerks diminished.   |
| <i>Florand.</i>   |      |      |       |   |           |   |
| 82                | m.*  | ?    | Bil.  | Obesity, rheumatism.  | 10        | Prolonged standing causes intolerable pain. Disappeared as a result of treatment for the obesity.   |
| <i>Le Gendre.</i> |      |      |       |   |           |   |
| 83                | m.   | ?    | R.    | Injury, gout.   | 20        | Paroxysmal attacks. Area of anesthesia. Paresthesia in other parts of the body.   |
| <i>Dalché.</i>    |      |      |       |   |           |   |
| 84                | f.   | 35   | R.    | Pregnancy.  | 4 mo.     | Hypesthesia. Cured by delivery.   |
| <i>Rapin.</i>     |      |      |       |   |           |   |
| 85                | m.*  | 62   | R.    | ?   | 25        | Paresthetic and anesthetic region does not show goose flesh upon applications of cold.  |
| <i>Féré.</i>      |      |      |       |   |           |   |
| 86                | m.   | 52   | ?     | Father alcoholic, epileptic; arrested development by typhoid fever. Repeated attacks of herpes preputialis. | ?         | Attacks of meralgia during the herpetic eruptions. Hyperesthesia, pain limited to anterior region of thigh.   |



|                             | Sex. | Age. | Side. | Cause.   | Duration. | Remarks.   |
|-----------------------------|------|------|-------|--|-----------|--|
| <i>Cora.</i>                |      |      |       |  |           |  |
| 87                          | m.*  | 50   | R.    | Sunstroke, sciatica.   | 7 mo.     | Hypesthesia to all forms of sensation, loss of power; paresthesia, worse in a warm bed.                |
| <i>Venturi.</i>             |      |      |       |  |           |  |
| 88                          | m.   | 44   | R.    | Syphilitic, at time of infection 20 years ago, had sensation of moisture on inner side of thigh. | 3         | Slight tactile hypesthesia only.   |
| 89                          | m.   | 62   | R.    | Corpulent.   | 20        | Sensation of cold after long sitting; no disturbance of sensation.                                     |
| <i>Musser &amp; Sailer.</i> |      |      |       |  |           |  |
| 90                          | m.   | 28   | Bil.  | Typhoid fever, catarrhal jaundice, erysipelas, possibly traumatism.                              | 1         | Complete cure, paresthesia. All sensation diminished. Tender nerve trunks.                             |
| 91                          | m.   | 30   | R.    | Gouty diathesis. Hereditary influence.   | 3         | All forms of sensation diminished, including electro-cutaneous. Nerve trunks not tender.               |
| 92                          | m.   | 76   | L.    | Gout and eczema.   | 16        | Hyperesthesia. Worse by exercise or stormy weather.  |
| 93                          | f.   | 41   | Bil.  | Gouty and neurotic diathesis.  |           | Attacks coming on during menstruation.   |
| 94                          | f.   | 24   | L.    | Pregnancy.   | 2         | Paresthesia and hyperalgia to pressure.  |
| 95                          | f.   | 22   | L.    | Successive attacks of eczema. Rheumatism. Hereditary influence.                                  | 2         | The attack commenced suddenly and left persistent numbness.  |
| 96                          | f.   | 23   | Bil.  | Parturition and menstruation.  | 3         | No disturbance of sensation.   |
| 97                          | m.   | 50   | R.    | Walking, exposure to cold.   | 4         | Complete anesthesia and analgesia, with smaller area in left leg. Nerve trunks not tender in left leg. |
| 98                          | m.*  | 60   | R.    | Influenza.   | 7         | Sensation of moisture.   |
| 99                          | m.   | 55   | L.    | Typhoid pneumonia.   | 13        | Hypalgia on external and lower anterior third of the thigh. Tactile sensation normal.                  |

The analysis of the statistics in the accompanying table gives the following results: In regard to the age, it is not given in 16 cases; in 2 it is stated that the patients were young. Between the years of 10 and 20, there was 1 case; between 20 and 30, 11 cases; between 30 and 40, 19 cases; between 40 and 50, 20 cases; between 50 and 60, 18 cases; between 60 and 70, 9 cases, and above 70, 2 cases. It must be noted that the age in

\* Physician.

R—right side. L—left side. Bil.—both sides. The numerals in the fifth column refer to years, unless otherwise stated.

all cases is that of the time of observation, and in many instances the disease commenced many years before. It appears that the disease apparently is far more frequent between the ages of 30 and 60, although the statistics of our own cases are not in accordance with this. In regard to the sex, there were 75 males, 21 females, and in 3 it was not given. Again our results are in slight disaccord with those of the majority of observers. The distribution of the symptoms in regard to the side is as follows: Not given, 16; on the right thigh only, 32; on the left thigh only, 31; bilateral, 20. The duration of the disease cannot be satisfactorily determined from the above tables. The majority of cases were reported before any improvement had taken place. In some cases the symptoms recurred at long intervals, and in many, the disease commenced insidiously, and it could not be determined how long it had existed. The etiology is not given with entire satisfaction. In many instances it is possible that the patient withheld important data, and some of the cases were simply reported on account of their symptomatology. The following points, however, can be determined: in all cases, including our own—injury is mentioned 19 times, syphilis 10 times, alcoholism 8 times, typhoid fever 8 times, and other infectious processes 8 times. Pregnancy appears to have been responsible for the development of the symptoms in 6 cases. Other nervous conditions were present in 11 cases, including 2 cases of locomotor ataxia. Among the less frequent causes were cold douches in 3 instances, obesity in 2, direct inheritance in 2, lead-poisoning in 1, exposure to heat in 1, and exposure to cold in 7. Gout and rheumatism were stated to have been present in 12 cases. Ten of the patients were physicians, an unduly large proportion, that is possibly to be explained by their more careful observance of their symptoms.

## REFERENCES.

- Adler, *Neurologisches Centralblatt*, 1897, p. 682.  
Bacelli, *Académie de Méd. de Gènes*. Quoted by Sabrazes et Cabannes, *de méd.*, 1897, p. 854.  
Bellot, *Journ. de médecine et de chirurgie pratique*, 1895, p. 683.  
Bernhardt, *Neurologisches Centralblatt*, 1895, p. 242.  
Bernhardt, *Revue neurologique*, 1895, p. 642.  
Bernhardt, *Deutsch. Archiv f. klin. Med.*, Vol. 22, p. 362, 1878.  
Bernhardt, "Krankheiten der peripherischen Nerven," Vienna, 1895.  
Bieganski, quoted by Goldflam.  
Charcot, *Leçons de Mardi*, 1887-8, Paris, 1892, p. 44.

- Cora, *Gazetta degli ospedali e delle cliniche*, Oct. 30, '98, p. 1369.  
 Claisse, *Gaz. des hôpitaux*, Paris, 1898, LXXI, p. 1377 to p. 1379.  
 Claisse, *Bull. et memoirs de la société médicale des hôpitaux de Paris*, 1898; third series, XV, p. 901 to 906.  
 Dalché, in discussion of Claisse's paper; *vide supra*.  
 Devic, *Province médicale*, No. 26, 1895. Quoted in *Journ. de médecine et de chirurgie pratique*, 1895, p. 529.  
 Donath, *Wiener med. Woch.*, 1897, p. 25.  
 Dopfer "Des troubles de la sensibilité dans la zone du nerf femoro-cutané," *Lyon*, 1896.  
 Escat, *Revue neurologique*, 1895, No. 20.  
 Féré, *Médecine moderne*, Paris, 1898, p. 49.  
 Freund, *Neurologisches Centralblatt*, 1895, p. 491.  
 Florand, in discussion of Claisse's paper; *vide supra*.  
 Goldflam, *Deutsche medicinische Wochenschrift*, 1895, p. 587.  
 Good, *Neurologisches Centralblatt*, 1898, p. 57.  
 Hager, *Deutsche med. Woch.*, 1885, p. 218.  
 Hirsch, *Journ. of Nervous and Mental Disease*, 1895, p. 193.  
 Ingelrans, *Echo médicale du Nord*, Feb. 6, 1898. Abst. in *Revue neurologique*, 1898, p. 253.  
 Kalischer, *Allg. medizinische Zeitung*, Vol. 65, No. 48.  
 Knauer, *Centralblatt f. Nervenheilkunde u. Psychiatrie*, 1896, p. 297.  
 Köster, *Neurologisches Centralblatt*, 1897, p. 252.  
 Le Gendre, in discussion of Claisse's paper.  
 v. Lutzenberger, *Neurologisches Centralblatt*, 1896, p. 1026.  
 Lucas-Champonnière, *Journ. de médecine et de chirurgie pratique*, 1895, p. 536.  
 Lop, *La Presse médicale*, March 1, 1898, p. 101.  
 Möbius, *Schmidt's Jahrbücher*, Vol. 247.  
 Nücke, *Neurologisches Centralblatt*, 1895, p. 338.  
 Nücke, *Rivista quindicinale di psicologia e di psichiatria*, 1897. Author's abstract, *Neurologisches Centralblatt*, 1898, p. 77.  
 v. Nartowski, *Neurologisches Centralblatt*, 1898, p. 1082.  
 Nawratski, *Centralblatt f. Nervenheilkunde u. Psychiatrie*, 1899, p. 109.  
 Osler, *Journ. of Nervous and Mental Disease*, 1897.  
 Pieracini, *La settimana medica dello sperimentale*, 1896, p. 371.  
 Pitres, quoted by Sabrazes and Cabannes, *loc. cit*.  
 Rapin, quoted by Bernhardt, "Krankheiten der peripherischen Nerven," *Vienna*, 1895.  
 Roth, "Meralgia paræsthetica," *Berlin*, 1895.  
 Sabrazes and Cabannes, *Revue de méd.*, 1897, p. 782.  
 Shaw, *New York Medical Journ.*, Feb. 13, 1897.  
 Spiller, *Journ. of Nervous and Mental Disease*, 1898, p. 756.  
 Souques, *Revue neurologique*, Aug. 30, 1899, p. 530.  
 Stembo, *St. Petersberger med. Woch.*, 1896, No. 20.  
 Traugott, *Monatschrift f. Psychiatrie u. Neurologie*, Vol. 3, 1898.  
 Venturi, *La riforma medica*, Feb. 17, '96, p. 460.  
 Verhoogen, *Journ. médicale de Brussels*, April 22, 29, 1897.  
 Warda, *Neurologisches Centralblatt*, 1897, p. 948.

References have been found to the following articles on this subject, but it was impossible to obtain the journals in which they occurred, and they are merely added for the sake of completing the bibliography:

- Beduschi, *Tribuna medica*, Milan, 1896, II, p. 96-98.  
 Claus, *Med. Weekbl.*, Amsterdam, 1895-6 II, p. 499 to 501.

- Dethlefsen, Ugesk. f. Laeger, Copenhagen, 1896, 5, R.  
 Haskovec, Lek. Rozledg., Prague, 1899, VII, p. 1 to 11.  
 Joncheray, Arch. méd. d'Angers, 1899, II, p. 514 to 518.  
 Roth, Med. Obozr., Moscow, 1895, XLIII, p. 678 to 688.  
 Dertot, Province médicale, 1895, p. 331.  
 Möller, Allg. med. Centralzeitung, 1896, p. 826.

2. ON SOME CASES OF PARALYSIS OF THE SERRATUS MAGNUS. Robert Jones, and G. P. Newboldt (The Liverpool Medico-Chirurgical Journal, 37, July, 1899, p. 482).

Paralysis of the serratus magnus does not occur very frequently. The writers of this article, after reviewing the anatomy, nerve supply, and actions of this muscle, present five cases of its paralysis. The most common cause of the paralysis, as reported by various observers, which are cited, is trauma in the neck to the posterior thoracic nerve. There have been cases due to a strain of the arm, to severe muscular effort, to long continued exertion, to carrying heavy weights upon the shoulder, and also to a neuritis following parturition, the neuritis excited probably by the muscular exertion or perhaps by an exposure of the neck to cold during the labor. In one case the nerve was imbedded in indurated tissue accompanying some inflamed cervical glands. Cases are recorded in which it followed typhoid fever. It may come on after gunshot wounds, and tumors in the neck may produce it by pressure on the nerve. The paralysis occurs more frequently on the right side, and between the ages of twenty and forty. It is rarely bilateral; one such case is reported as having occurred after pertussis. Males are affected more often than females. Of the five cases presented in this article, however, four were females. Two cases gave a history of injury or strain; in the third case the paralysis preceded and in the fourth followed parturition, and one case, that of the man, was probably of rheumatic origin.

In four of the five cases the right side was affected.

The three most prominent symptoms of paralysis of this muscle were found to be—inability to raise the arm above the right angle; inability to reach forward as compared with the opposite side, and the projection of the scapula as a wing, on the affected side, when the patient raises the arm and holds it directly forward. The most marked of these three symptoms, and present in all five cases, was the inability to raise the arm above a right angle. One patient complained first of his inability to reach the ink-stand when at his desk. All these cases could elevate the arm when the shoulder blade was pressed to the side. Bruns, however, cites a case of paralysis of the serratus magnus in which this symptom was absent and the arm could be elevated. The expansion of the unaffected was greater than that of the affected side, on deep inspiration with the arms raised, in the one case examined on this point. Pain was present in some of the cases. When the arms hung by the side of the body, the scapula of the affected side was slightly higher and nearer the spine than that of the sound side, and there was some obliquity of its inner border.

Rough massage and muscle beating, together with a sling to support and raise the arm and shoulder, was the treatment given.

All were improving, or had recovered, under this treatment.

The prognosis is good if treatment is begun early and persevered in, but progress is slow. Some good photographs show the projecting scapulæ and limitation of movement of the arms. BONAR.

## THE UNITY OF THE ACUTE PSYCHOSES.<sup>1</sup>

BY PHILIP COOMBS KNAPP, M.D.

Dr. Knapp stated that there were several forms of melancholia, differing so much in type as to suggest different diseases. Melancholy seemed rather a symptom than a disease by itself. The existence of mania, acute paranoia, and many other acute mental diseases was questioned, most writers disagreeing in their classification. A symptomatic classification of mental diseases was often fallacious and unsatisfactory. Alcohol might cause a number of different types of psychosis. The acute psychoses were often of toxic origin; they might attack healthy persons, they might cause death by exhaustion, recover completely, or end in dementia or chronic delusions. They were regarded as probably one affection, under the general heading of Meynert's amentia.

### DISCUSSION.

Dr. C. K. Mills said that it seemed to him that much in this paper may mean a step backward in our methods of looking at these diseases. He may not have fully understood the author, but if he intended to put together—that is, in the same class—diseases so distinct clinically and etiologically as paranoia, melancholia and mania, he had not firm ground upon which to stand. One point in the paper, that of the unity of certain acute psychoses, in a general way may be admitted, and it is by no means a new thought. It has long been discussed as to whether or not acute mania and acute melancholia, and acute delirium, are not diseases having the same method of origin, the same pathogenesis.

There is no doubt that the forms of melancholia, paranoia, and mania are at times too much elaborated, but the clinical distinctions between these affections are as certain to-day as they ever were. How Dr. Knapp separated these disorders for the purpose of treatment, unless upon the clinical basis with which we are so familiar, he did not know. Paranoia is a distinct disease from melancholia, has a different origin, course, and usually a different termination. Because some cases of melancholia pass to dementia, and because all cases of paranoia

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<sup>1</sup>Read at the twenty-fifth annual meeting of the American Neurological Association. June 14 and 15, 1899.

do so, is no reason why they should be regarded as the same disease. The illustrations Dr. Knapp gave concerning diphtheria, etc., are familiar to all, but they do not support the radical position taken. Hysteria, neurasthenia, melancholia, mania, and dementia paralytica can be as clearly separated as any set of diseases we have outside the domain of psychology.

With those authors who discuss these disorders from the standpoint of large experience, he believed the tendency to be in an opposite direction to that manifested by Dr. Knapp.

Dr. F. X. Dercum agreed with Dr. Mills. He said the facts warrant no other conclusion than that the various well-recognized forms of mental disease are distinct clinical entities. He further said that because a patient who develops melancholia passes through a series of vague symptoms, superficially resembling neurasthenia, we are not justified in maintaining that neurasthenia and melancholia are the same disease. The period of evolution of melancholia differs essentially from the syndrome known as neurasthenia. He suggested that to the symptoms of the period of evolution should be applied the term *neurasthenoid*, for it is never a true neurasthenic condition. Similarly, it is improper to apply the term *neurasthenic* to the prodromal period of paranoia, a period of slow evolution often covering years. Dr. Dercum, with Dr. Mills, regarded Dr. Knapp's attitude as a retrograde one. He did not think it wise to cast aside the clinical distinctions which enable us to make reasonably safe prognostications, and to indicate more or less successful plans of treatment.

Dr. H. T. Patrick thought Dr. Knapp had been entertaining the association, and himself, with a speculative pathology, and if we realized that we might meet on quite common ground in the discussion. There can be no objection to putting all the acute psychoses into one general class on the strength of similarity of symptoms, but when Dr. Knapp does that he has as indefinite an entity as the New England conscience of which he spoke. Dr. Patrick agreed with Dr. Mills that in adopting such a standard we should be taking a step backward. The tendency is, and ought to be, toward a more refined differentiation between these diseases, because this will help us in the treatment. It is true that acute alcoholism may be "bellicose, jocose, lachrymose," etc., but still it is an acute alcoholism, it must be recognized as of one type or another, and it must be recognized as different from chronic alcoholism. So with the acute psychoses. There may be certain similarities, and we may not on one day be able to tell which of these conditions it is, still they are distinct, and should be so regarded.

Dr. H. Upson remarked that in dealing with such a ques-

tion as this we are groping in the dark, until we can find out the causes of these acute psychoses. He did not believe that acute melancholia and acute mania are pure psychoses, but that these diseases have a definite chemical basis that probably will be proven ultimately to be toxic, and probably back of that to be due to bacteria; meanwhile, the best thing we can do is to make our classification from the symptom-complex.

Dr. Joseph Collins said that if he had heard Dr. Knapp correctly, it seemed to him that those who honored him by this discussion had, in a measure at least, misunderstood his position. Although Dr. Collins was convinced, from a purely utilitarian point of view, of the unadvisability of taking the position Dr. Knapp did, yet he saw that Dr. Knapp could defend himself logically. It was merely good logic put to a bad purpose. If one so desires there is no objection to considering all forms of insanity, acute, chronic, inherited, or what not, as of a similar nature. They are all dependent upon inherited or acquired functional instability of those brain cells subserving intellectual activity, which in turn may be simply a nutritional condition. If one wishes on these grounds to contend for the unity of the acute psychoses no one can gainsay him the contention. It has been definitely proven that in many forms of insanity there is some degeneration of the neurons. The dementia following attacks of acute mania and melancholia, and preceding recovery, is very likely the expression of profound chromatolysis in the cell body and of changes in the dendrites, while recovery is the expression of recuperation and reconstruction in these components of the neurons. In all of the acute psychoses it is highly probable that the anatomical change is one of degree rather than of kind. When our methods of investigation become as keen as our pursuit of them, we shall probably find just what these changes are. But even then it will not assist us in our recognition and handling of these conditions, and it is from this point of view particularly that Dr. Knapp's effort was a retrograde step. For instance, it has not facilitated the recognition of acute hemorrhagic encephalitis to know that it is of the same causation and pathogenesis as anterior poliomyelitis, nor has it furthered our efforts in shaping the course of this disease. It has been the labor of several generations to differentiate the forms of insanity clinically, in order that we may recognize them readily and apply the treatment that we know from personal and inherited experience is beneficial. What does it avail to know that pathogenetically there is an unity of acute delirium and acute hysterical or acute delusional insanity? Does it help us any in the treatment of these conditions? It does not. If we were to attempt to propagate Dr. Knapp's ideas

among beginners in psychiatry there would be a flood of obscurity let in upon them that their descendants would scarcely emerge from. It is entirely from this utilitarian standpoint that Dr. Collins objected to the propagation of Dr. Knapp's doctrines. He did not impugn his logic, but his premises.

Dr. J. W. Putnam said that in listening to the arguments he had been interested in comparing them with his experience in various etiological factors in acute psychosis, and two or three cases that he had seen following operations during which the carotid artery had been tied came to his mind. He had seen mania develop under such conditions, and, knowing the previous state of the patient, was enabled to state what the prognosis would probably be. The mania was as wild and the movements as active and purposeless as are seen in any form of mania, but knowing that the mania had been due to a sudden disturbance of the cerebral circulation, it was easy to show that it was the result of shock. It seemed to Dr. Putnam, in classifying such cases as these, to say that there was a unity between them and one of periodical or puerperal mania would require a stretch of imagination. We have all seen persons in apparently good health who receive a shock to the nervous system as the result of fear, joy, or grief, and a change of mental action result so great as to develop either melancholia or mania. Dr. Putnam thought that it is true that you cannot tell what type of mental change is going to result from a given etiological factor; as the result of fright you may get acute dementia, melancholia, or mania. He had no doubt Dr. Knapp himself would make his prognosis depend largely upon the etiological factor, and according as that changes would give a serious prognosis in one case, while in another he would assure the persons concerned that the affair would be but temporary.

Dr. Collins, in reference to the statement made by Dr. Putnam, that after tying the carotid artery an acute melancholia may develop, which, according to Dr. Knapp's idea, is due to a degeneration of the neurons, wished to know whether it takes a very great stretch of the imagination to compare a myelitis that results from a disturbance of the circulation due to tying the abdominal aorta, to the myelitis that occurs with puerperal fever.

Dr. W. G. Spiller said that Dr. Knapp was attempting to unite the acute psychoses from their clinical appearances, while Dr. Collins sought an anatomical basis, which at present is most uncertain. Dr. Knapp had not attempted to classify mental diseases from their pathological changes, and in the light of our present knowledge such an attempt must utterly fail.

Dr. Spiller was not inclined to accept the statement made



by one of the speakers, that most neurologists believe that *all* acute diseases of the gray matter of the cord are the result of intoxication and infection. Many probably are, but diseases occur that are not produced by intoxication or infection. Softening of the cord probably may occur from closure of the blood vessels, and experiments have fully established the truth of this statement.

Dr. P. C. Knapp thought that the eloquent attacks upon his position by Dr. Mills and Dr. Dercum were due to a misconception. He may have taken a step backward, but he did not think he went so far backward as to adopt the text-book that Dr. Mills had cited as an authority. He thought that a glance at the text-books of Kraepelin, Ziehen, Schüle, Krafft-Ebing, Agostini, or Morselli, would show that the acute psychoses described by those writers vary very materially. As he said in his paper, Schüle and Aschaffenburg are contesting about the existence of catatonia. Ziehen upholds an acute paranoia, and Kraepelin says it cannot exist. Wernicke seems to have adopted practically the same position that Dr. Knapp had taken in maintaining that most of the acute psychoses are to be classed as Meynert's amentia, whose exhaustive chapter on that subject Dr. Knapp commended to Dr. Mills. Dr. Mills also implied that practical clinical experience would lead Dr. Knapp to take a different view. Dr. Knapp would admit that his experience might not be so great as Dr. Mills', but at the same time he had for a number of years been putting these ideas as to the acute psychoses into practical application in the study of actual cases of mental disease in its advanced forms, as seen in the asylums and in the border-land types in both asylum and private practice. Dr. Mills also misunderstood him in thinking that he would place paranoia and general paralysis in the same group with acute psychoses. He did not attempt to classify, and he should not think of classifying, typical paranoia or general paralysis of the insane with the acute mental disturbances, such as acute mania, acute melancholia, acute confusional insanity or catatonia. What he was speaking of was the similarity of the acute psychoses.

Dr. Knapp said that Dr. Patrick did not understand the New England conscience, because it was less common outside of New England, but he could assure him that it was a troublesome thing to deal with, and where it exists it makes the prognosis in neurasthenia much graver.

He agreed with Dr. Spiller that we cannot classify our cases on an absolute anatomical basis as yet, and that if we were given a definite lesion we could not predicate the mental disturbance that would follow, nor *vice versa*.

Dr. Knapp remarked that Dr. Collins had said he could not impugn his arguments, and yet he disagreed with him entirely; that seemed to Dr. Knapp a peculiar position to take, for if his arguments were sound the position must be accepted.

After all, what is the object of differentiation? It is made chiefly with regard to prognosis and treatment, and both of these must, after all, depend upon a study of the individual case. We see a thousand cases of typhoid fever, and if we have that clinical knowledge and experience that our President laid stress upon in his address, we form our prognosis in the individual case upon the individual symptoms that we know may determine whether the result will be good or bad. We make our prognosis according to the severity of the attack, and to its whole clinical aspect, but we regard the whole number of cases as one disease, though our prognosis depends upon different symptoms in the individual cases. We would not treat every case of typhoid fever in the same way, and we need not treat every case of mental disease in the same way, simply because they belong to a given class.

3. WEITERE BEITRÄGE ZUR LEHRE VON DEN CUTANEN NEUROTROPHISCHEN STÖRUNGEN (Further Contributions to the Subject of Neurotrophic Cutaneous Disturbances). Löwenfeld (Münchener medicinische Wochenschrift, 1899, Nos. 26 and 27).

The author reports the following cases: 1. "Neuritic flat hand." In this case there was a swelling and thickening of the tissues in the volar region of the left hand, in the ulnar distribution, the natural arched contour of the palm being obliterated, but without change of color or local lesion of the skin. Paresthesia and other evidences of neuritis were present.

The case occurred in a woman of middle age of rheumatic diathesis.

2. "Neuritic flat foot," also in a female of middle age, who had had glycosuria and was probably of arthritic diathesis.

In this case there was swelling and distortion of the sole, first of the right, then of the left, and then again of the right foot, with symptoms of neuritis of the corresponding nerves. Recovery eventually occurred in both cases.

3. "A peculiar cutaneous neurosis." This occurred in a woman of 37, of strongly neuropathic heredity, and who herself had presented symptoms of neurasthenia and hysteria. The author makes no mention of the presence or absence of stigmata.

In this patient, putting the hands into cold water rapidly produced great swelling of the fingers, with paresthesia. The skin over the affected area was pale, the swelling so great that the fingers could not be moved. The patient stated that at times after having been thoroughly chilled, she passed very dark urine. A specimen of such urine brought by the patient was examined by the author, and found to contain albumin, but no blood corpuscles or casts. After long continued use of electricity the sensitiveness to cold water gradually disappeared.

ALLEN.

## TWO CASES OF MUSCULAR DYSTROPHY WITH NECROPSY.

(One of the Facio-Scapulo-Humeral Type Reported Clinically  
by Duchenne, of Boulogne ; Landouzy and Dejerine).<sup>1</sup>

By WILLIAM G. SPILLER, M.D.

### ABSTRACT.

The first case was published by Duchenne, of Boulogne, in 1872, when the patient was nine years old, and later by Landouzy and Dejerine—also as a clinical observation—when he was twenty-eight years of age. The man died in the service of Dr. Dejerine at the Bicêtre Hospital, in Paris, about six years after the second clinical observation was published. The pathological material was given by Dr. Dejerine to Dr. Spiller for microscopical study.

The case was one of hereditary progressive muscular atrophy, beginning in the face when the patient was three years old. The atrophy was most pronounced in the face, shoulders and upper limbs. Permanent contractures existed in the extremities, but there were no pains, no fibrillary contractions, no hypertrophy, and no reaction of degeneration. At the necropsy the muscles were examined individually by Dr. Dejerine, and the atrophy was found to be intense. While many of the muscles of the face were greatly affected, the muscles of mastication and of the eyeballs were remarkably well preserved. The examination of all the muscles in detail will be published later. The intramuscular branches of the facial nerve, examined in the fresh state by Dr. Dejerine, and stained with osmic acid, appeared to be normal. The anterior and posterior roots, the nerves of certain muscles of the extremities, examined in the same way, were found to be normal. The nervous system studied by Dr. Spiller was found to be normal. Notwithstanding the intense atrophy of the muscles supplied by the seventh nerve, *the intramedullary portion of the facial nerve was perfectly normal on each side. The cells of the facial nucleus were large, had well-defined processes and centrally situated nuclei.*

<sup>1</sup>Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

This paper will form a part of the volume soon to be issued from the William Pepper Laboratory of Clinical Medicine.

The diaphragm was found greatly degenerated, but its intramuscular nerve bundles were normal. Although the deltoid muscle was merely a mass of connective tissue, the muscle spindles and intramuscular nerve fibers appeared to be normal.

The second case was that of a man who was in the clinic of Dr. Dejerine, at the Bicêtre. In this case the face was intact. The paralysis began quite acutely in the lower limbs, as the pseudohypertrophic form, when the boy was eight years old, and extended to the trunk and upper limbs, and the patient became almost completely paralyzed in the trunk and extremities. He died at the age of twenty. The muscles were found to be intensely atrophied, but the central and peripheral nervous systems were normal. In some sections of the spinal cord a few motor cells, which might be regarded as somewhat atrophied, were seen in the anterior horns.

Dr. Spiller gave a brief review of the most important literature on the Landouzy-Dejerine form of muscular dystrophy. He spoke especially of the atrophy of the diaphragm, showing that muscles believed at one time to be spared in the atrophic process may be involved; he spoke also of the presence of normal nerve fibers and normal muscle spindles within intensely degenerated muscular tissue, as showing that the disease is probably primarily muscular. He showed that muscular atrophy does not essentially differ in the various morbid processes, and that no histological findings can be regarded as absolutely pathognomonic of any form of muscular atrophy. He spoke of the great difficulty in some cases of making a clinical diagnosis in regard to the form of atrophy, and referred to recent papers in which this difficulty is so clearly illustrated. He called attention to the bulbar form of muscular dystrophy (Hoffmann) and to the infantile family form of bulbar paralysis of Fazio and Londe, in relation to the Landouzy-Dejerine type of muscular dystrophy. The cases of congenital facial paralysis reported by H. M. Thomas were also of difficult interpretation.

Dr. Spiller then discussed the subject of muscular dystrophy from a pathological standpoint, and referred to the cases in which the nervous system was found diseased. He gave as his conclusions:

1. Cases of muscular atrophy occur which present the well-known localizations regarded as characteristic of the different forms of muscular dystrophy. In the large majority of these cases the spinal cord and peripheral nerves are normal.

2. In other rare cases presenting the clinical features of muscular dystrophy, the nervous system is more or less involved.

3. The histologic changes in the muscles are not pathognomonic of any form of atrophy.

4. It is proper to regard muscular dystrophy as a disease usually distinct from spinal muscular atrophy, but transitional forms connect the myopathic and myelopathic types of atrophy.

#### DISCUSSION.

Dr. H. M. Thomas said in regard to the cases he reported last year, that when he first saw them the thought of their being juvenile muscular dystrophy occurred to him at once, and the reason he did not so consider them was, in the first place, that the histories as definitely as they could be gotten showed the condition to be congenital. It was noticed directly after birth. Then the distribution of the affected muscles was exactly the same as in the cases of congenital unilateral facial paralysis combined with affection of the ocular muscles. Then too, electrical examination of the affected muscles gave no response to any amount of current, while the other muscles in the facial group gave the normal response. These symptoms, together with dulness of hearing, which seemed to be congenital, and the fact that each had congenital abnormalities of the external ear, seemed to make the diagnosis pretty clear. They may later develop muscular dystrophy, but Dr. Thomas did not believe they would. He did not see why they should be thrown out because they were bilateral when they correspond in other respects to the congenital cases. Dr. Thomas said that the whole subject of progressive muscular dystrophy is, as Dr. Spiller had shown, extremely confusing, and his conclusions expressed the belief that Dr. Thomas had come to from reviewing the literature. There have been a good many articles written upon this subject, trying to bring together all the cases of muscular dystrophy and atrophy, but Dr. Thomas believed we shall have to wait for further investigations before we can do anything more than Dr. Spiller had done in his conclusions.

Dr. Thomas did not mean to say in his paper that in any case of congenital facial paralysis the muscles of the face had been examined. The cases he referred to in which a muscle examination had been made were cases of congenital eye-muscle defects.

## NEW YORK NEUROLOGICAL SOCIETY.

November 7, 1899.

The President, Dr. Frederick Peterson, in the chair.

### MULTIPLE NEURITIS AND ACROMEGALY.

Dr. William Hirsch presented a man, twenty-six years of age, who had come to his clinic on July 14, 1899, complaining of shooting pains in the upper and lower extremities, and great difficulty in walking. He stated that seven years ago he had had a similar attack of pain in the upper part of his body, and had been paralyzed for six months. Two years later he had had a similar attack, lasting ten weeks, and still later had had a third attack, though of shorter duration. Examination revealed very marked atrophy of the muscles of the upper extremity, especially the muscles about the shoulder. The long muscles of the back were also considerably atrophied, as were those in the lower extremities, but not to the same degree. Sensation was slightly diminished in the shoulder girdle. The motor power had been greatly decreased so that the man could walk only with the greatest difficulty. The pupils were equal and of normal reaction. The patellar reflexes were considerably exaggerated, but no ankle clonus was present. The case was evidently one of recurrent multiple neuritis, but in addition there was a distinct swelling of the upper lobe of the thyroid gland, and the hands and feet were disproportionately large, and more or less disfigured. The bones of the carpus and metacarpus were decidedly thickened at their ends. The internal and external condyles of the femur were much thickened, and the lower jaw was not of the usual shape. The heart was enlarged, and there were systolic and diastolic murmurs at the apex. The patient said that he had noticed a gradual enlargement of the hands and feet since the age of twenty-one, and he had grown about two inches in height during the last few years. A diagnosis of acromegaly was made. Plaster casts of the hands and feet were taken. The man was placed on the extract of hypophysis. He recovered from his neuritis in about two months, so that he could now walk perfectly well. Such marked diminution in the size of the hands during this period had occurred that casts made two days previously were also presented to allow of comparison with those first taken. Skiagraphs showed that the chief changes had been in the soft tissues. The speaker said that the recurrence of multiple neuritis seemed to be quite in harmony

with the theory that acromegaly was the result of a disturbance of metabolism from disease of the hypophysis.

Dr. W. M. Leszynsky thought that the case was an atypical one, and he would not be willing to accept the diagnosis until a careful examination for spinal curvature had been made and the visual fields had been tested.

Dr. Terriberry asked what Dr. Hirsch considered to be the pathological condition giving rise to the neuritis. The existence of fibrous enlargement of the joints pointed rather to a neuritis of rheumatic origin.

Dr. Joseph Collins was also inclined to think that the case was one of rheumatic multiple neuritis. Acromegaly, he said, was really a uniform enlargement of the joints—a condition which the photographs and skiagraphs showed did not exist here. The hands and feet were misshapen, as might occur in rheumatic arthritis. The condition of the heart also favored the view that the case was rheumatic.

Dr. B. Sachs said that, while the case was certainly atypical, the increase in stature in recent years and the peculiar shape of the lower jaw seemed to him to point rather strongly to acromegaly. The condition was not a thickening of the lower jaw, but an actual prolongation of the ramus of the jaw. The diagnosis of a rheumatic affection did not entirely cover all of the facts in the case.

Dr. Hirsch said that, in his opinion, the skiagraphs showed clearly that the joints themselves were perfectly normal, although the ends of the bones were somewhat thickened. That in itself should exclude joint disease. Again, the marked swelling of the thyroid was not compatible with the rheumatic theory. The increase in the lateral diameter of the foot was the most characteristic change of all. As the nerves were involved in acromegaly at times, it was quite proper to look upon the neuritis as having the same origin. As only about one hundred cases of acromegaly in all had been reported, one was hardly warranted in speaking of the disease as being "typical."

#### AMAUROTIC FAMILY IDIOCY.

Dr. F. Peterson presented a child, fifteen months old, the only child of a Hebrew mother and born when the mother was nineteen years of age. The pregnancy had been normal and the labor difficult and instrumental. Slight asphyxia had occurred at birth. The child had been nursed until two months ago. It had been well up to the age of five months when it had had some fever, and shortly afterward had developed a very painful condition of the cervical spine. The case had been sent to the nervous department of the Vanderbilt Clinic by Dr. Gibney. The arms and legs had been somewhat rigid; the head muscles also rigid. The child was nearly blind, and the disk showed a typical white atrophy with the cherry red spots. The strange feature of the case was its apparent commencement as a meningitis.

Dr. V. P. Gibney stated that he had examined the child and had failed to find any condition demanding orthopedic surgery.

Dr. Terriberry said that he had obtained a history of the child having been severely injured by instruments at birth, and the mother had called attention to a scar in the frontal region, and had stated that the attending physician had thought this injury so severe that he had

doubted if the child would survive. According to his recollection, the mother had told him that the trouble had begun much sooner after birth than had been stated in the history just given.

Dr. Sachs said that there was no reason why a child with amaurotic family idiocy should not have an acute cerebral disturbance, and hence, the acute symptoms detailed would not militate against the diagnosis. The ophthalmoscopic picture confirmed this view.

Dr. Hirsch regarded as worthy of note that there was still some vision remaining, as some authorities had reported cases of this kind in which the children had been born blind. He had examined one of these cases anatomically, and had come to the conclusion that he had had to deal with an acquired disease, and not with an arrest of development. The case just presented afforded additional support to this view.

Dr. Peterson said that he had carefully inquired into the history, and had been told by the father that the child had been apparently healthy up to the age of five months, or until the illness marked by fever and croupy cough. The marked retraction of the head and the great tenderness in this region had been so great as to lead a good pediatricist and a surgeon into the belief that there was some serious injury—probably a dislocation of the cervical vertebræ. Typical white atrophy was present. The child's general health was failing.

#### CASES OF RIGID SPINE.

Dr. Joseph Collins presented a man, forty-five years of age, who had served in the German army. About ten years ago the present trouble had gradually developed—the bending of the back and the difficulty in moving one hip. Examination had shown a cessation of abdominal respiration and a rigidity of the spine.

Dr. V. P. Gibney showed a similar case, that of a man, thirty-five years of age, who for the past five years had been stooping a good deal. About seventeen years ago he had had an attack of gonorrheal rheumatism lasting three or four weeks, and involving the lower extremities. Four years later he had had an attack of sciatica on the left side, lasting nearly a year. In October of 1896 much pain had been felt in the heel and it extended up into the hips.

Dr. B. Sachs and Dr. J. Fraenkel read a paper with the title: "Progressive ankylotic rigidity of the spine (spondylose rhizomélique)." See page 1.

Dr. Gibney said that his attention had been called to the subject about eighteen years ago by a case of spondylitis deformans, and since then he had been trying to differentiate the many varieties of stiff back coming under his observation. He had seen a number of cases of senile arthritis.

Dr. C. L. Dana said that he had been taught that there were two types of arthritis deformans, viz.: (1) The type beginning in the small joints and progressing steadily, and (2) the senile type, beginning in the back and hips and stiffening them. There were almost always some cases of that kind in Bellevue Hospital. He had studied his own cases in connection with those reported by others, and had come to the conclusion that the cases of the Strümpell-Marie type were nothing



more than arthritis deformans. He felt sure that in due time cases would be found in which the spine only was affected, and in which only the root joints were affected. He now had a case in which only the hips were affected, the spine not having been involved as yet. He did not personally feel that either Strümpell or Marie had added anything at all to the clinical knowledge of these cases. Twenty-five years ago it had been stated very clearly that rheumatoid arthritis sometimes presented this particular form. Von Bechterew had certainly described types that were distinctly secondary; the case reported with autopsy seemed to him one of specific meningitis. There was a type of stiff spine which had been described by Dr. Bradford, of Boston, and proved by him to be due to gonorrheal rheumatism. In this case the history was most conclusive. He now had under observation a very bold kyphotic spine, and the case appeared to him with little doubt as one of rheumatoid arthritis. He, therefore, believed that "spondylose rhizomélisque" was a nosological superfluity.

Dr. George R. Elliott said that three years ago he had been privileged to examine under ether, with Dr. Fraenkel, one of the patients presented this evening, and had found that the rigidity had remained. Since that time the case had proved to be a classical type of "spondylose rhizomélisque" of the Strümpell-Marie type. He was not yet willing to admit that we were dealing with a disease *per se*. That the case was not one of arthritis deformans admitted of little doubt. It had been said that this type could be differentiated from arthritis deformans in that the latter affected chiefly the small joints, but he believed such a statement must be modified. A clinical examination of so-called arthritis deformans revealed two distinct findings, viz.: one, an atrophic process, in which, after acute or subacute symptoms, the joint got smaller; and a second clinical picture, in which the joints became hypertrophied. Pathological study supported the clinical findings. He could see nothing in either the clinical history or in the pathological study of the type presented which differed from the hypertrophic form of the smaller joints. He was of the opinion that there were two distinct diseases—arthritis deformans and a proliferating arthritis. This might be called the Strümpell-Marie type, or better, central type of the proliferating kind, as distinguished from the peripheral type of the proliferating kind; but it was not to be confounded with arthritis deformans proper. That the small joints always escaped, as had at first been held, was not true. Marie himself had reported involvement of small joints in one of his cases. This statement was supported by Bannatine, of England, and Goldthwait, of Boston. There was much about the type of disease under discussion to suggest a degeneration, viz.: Gradual destruction of the soft parts of the joint and partial replacement by osseous material, the parts becoming welded together with very little excess of tissue or deformity. The proliferation, which was slight, appeared as a secondary process, the result of irritation. It was very unlike that producing so-called arthritis deformans proper. It was not so rare as might be supposed from the few cases so far reported. Dr. Goldthwait had reported at the last meeting of the American Orthopedic Association ten cases which had come under his observation. So marked a type as one of the cases just presented was rare indeed; it was a classical example of the Strümpell-Marie type, or central type.

Dr. W. R. Townsend said that he had at present under his care a child presenting a picture very closely resembling arthritis deformans. If he remembered correctly, Charcot had insisted that these cases were not rheumatic, but neurotic. He had seen a number of cases of rigid spine, and had supposed them to be either rheumatic or of the types

of arthritis deformans of the neurotic character described by Charcot.

Dr. Collins said that a discussion of this kind could not be profitably carried on until the various conditions spoken of had been clearly differentiated. He then reported two cases occurring in women. The first case was a woman of thirty years, first seen in the City Hospital last spring. She had been well up to the fall of 1897, at which time she had had what she called "a stroke on the left side." In August, 1898, she had begun to complain of pain in her back, and she soon became so stiff that she could not get around. Examination had shown over the sacrum what had at first appeared to be a bony tumor of the sacrum. No sensory disturbances of the legs and no trophic cutaneous disturbances had been seen, although she had been on her back for a long time. The right upper extremity was stiff at the shoulder joint. No evidence of cranial nerve palsy existed. At the autopsy the stiffness in the hip joints was noted to be less than during life. The liver showed marked passive hyperemia; the kidneys cloudy swelling, and the heart marked fibrous endocarditis and advanced myocarditis—a cartilaginous myocarditis—and ossification of the fibers and fatty degeneration. The brain and spinal cord had been stained and examined, but had appeared to be normal. The vertebral column seemed to be made of one piece, and this was due entirely to the calcification of the anterior spinal, of the posterior spinal, and the interspinal ligaments. The intervertebral disks were not thickened. The change in the hip joints was comparatively slight. The shoulder joint was not examined. The changes, therefore, were not those of an inflammation, and were not like those of an ordinary rheumatism. The speaker said that cervical meningitis would produce a stiff back and a stiff neck—a symptomatic stiff back—and it seemed to him this was the character of von Bechterew's case.

Dr. Peterson said that he had seen three or four cases, all of which he had diagnosed as arthritis deformans of rheumatic origin. One of these patients, a man of forty, was of a neurotic temperament, and had been previously under the care of a number of specialists. He had developed in the course of the preceding three or four years an almost complete immobility of the spine and stiffness of the hips and knees. No enlargement of the small joints of the hands and feet and no evidence of cardiac disease were detected. If cases of this type were excluded, as Dr. Collins had suggested, there would be very few left. What Dr. Collins had found at autopsy seemed very much like what had been observed in many cases of arthritis deformans of the rheumatic type.

Dr. Sachs, in closing, remarked that the case described by Dr. Collins seemed to be rather acute as compared with those that had been reported. The post-mortem findings corresponded closely with some von Bechterew had reported. The von Bechterew type had been brought into the discussion because it had been necessary to differentiate it from the others, and for this reason he had endeavored in the paper to show more clearly than previous writers that von Bechterew's case was of the secondary type. The morbid changes were quite different in the two types. In the European journals there had been much opposition to the view that they were at all closely allied to rheumatism or to arthritis deformans.

## Periscope.

### CLINICAL NEUROLOGY.

4. LE DIAGNOSTIC DIFFERENTIEL DE L'APOPLEXIE HYSTERIQUE ET DE L'APOPLEXIE ORGANIQUE (The Differential Diagnosis Between Hysterical Apoplexy and Organic Apoplexy). J. Crocq (*Journal de Neurologie*, No. 21, 1899, p. 410).

There are cases of apoplexy in which in the beginning of the "insult" it is impossible to say whether the affection is organic or hysterical. By apoplexy Crocq means a sudden loss of consciousness, sensation and motion, without much alteration of respiration or circulation. Crocq refers to a case previously reported by him. A hysterical woman of 24 years had an apoplectic attack; she remained forty-eight hours in coma, with stertorous respiration, congestion of the head, left hemiplegia, apparently paralysis in the lower part of the distribution of the right facial nerve, and paralysis of the tongue. The lower limb (left (?)) became contracted after about thirty-six hours. Consciousness returned after forty-eight hours and respiration became less difficult. Sensation returned in the sound side on the third day, but the paralyzed side remained anesthetic. Deglutition and speech were impossible, and pressure in the ovarian regions was painful. The symptoms disappeared as if by magic. Faradization given daily led to complete recovery within ten days. The case was at first believed to be one of crossed paralysis of pontile origin, but the diagnosis of hysterical apoplexy was believed by the third day to be positive on account of the rapid amelioration, the recognition of the facial condition as one of spasm, the anesthesia of the left side of the body with normal sensation in the face, the pain on pressure in the ovarian region, and the contraction of the lower limb.

Crocq reports another case in which the diagnosis was difficult. A woman of 50 years had had left hemiplegia for a year and a half, involving the left side of the face, without disturbance of sensation. The paralysis began with an apoplectic attack and contracture of the left lower limb developed. Faradization applied once caused the contracture to disappear, and the limb that had been immobile in flexion for a year could be moved with considerable power. How much of the patient's condition was organic and how much hysterical could not be determined.

He reports another case of apoplexy occurring suddenly in a hysterical man without any organic lesion being found at the necropsy.

SPILLER.

5. LE PRURIT ET LA TRICHOTILLOMANIE CHEZ LES PARALYTIQUES GENERAUX (Pruritus and Trichotillomania in General Paresis). Ch. Feré (*Nouvelle Iconographie de la Salpêtrière*, July and August, 1899, No. 4).

Lesions of the skin are by no means rare in general paralytics. The most frequent are the erythematous or bullous eruptions, purpura, herpes zoster. Outside of these, however, are the lesions of less severe character, which belong to the class of dermato-neuroses. The pruritus is less often found among general paralytics than is commonly supposed. In twenty-six cases, it was found only three times. Trichotillomania is defined by Hallopeau as an affection characterized by an intense pruritus in all the hairy parts of the body, and by a true vesania, which not only compels the patient to scratch, but to tear and pull away the hairs near their point of emergence from the skin. There results,

therefore, an artificial alopecia. Neither the skin nor the hair shows any pathological appearances whatever.

The case quoted to illustrate this condition is that of a general paralytic 37 years old, with the usual symptoms. Following an epileptiform attack, he lost considerable weight. Shortly afterwards, upon examination, a large part of the pubic and genital hair was found to be absent. A pruritus evidently existed. No lesion in either the skin or hair could be found. This condition continued for a few months, when it suddenly ceased, without any definite cause. The hair soon grew again.

Autointoxication was thought of as an explanation, but on account of the failure of other symptoms was disregarded. The only explanation is, that we have to do with the primary localized pruritus in a general paralytic.

SCHWAB.

6. EIN BEITRAG ZUR FRAGE NACH DER EXISTENZ VON NOTHNAGEL'S KRAMPFZENTRUM IN DER VAROLSBRÜCKE DES MENSCHEN (A Contribution to the Question of the Existence of Nothnagel's Convulsive Center in the Pons of Man). Hans Luce (*Deutsche Zeitschrift für Nervenheilkunde*, Vol. 15, Nos. 5 and 6, p. 327).

Whether or not irritation of the pons will produce true epileptic convulsions independent of the influence of the cerebral cortex is a question that has perplexed neurologists for years. Luce reports a case in which he observed chronic convulsions involving the whole musculature of the body, except the portions innervated by the sixth and seventh nerves. These general convulsions were believed to be the immediate result of an extensive pontile hemorrhage. Luce makes a careful study of the literature and concludes that pontile hemorrhages may cause general epileptic convulsions. These are produced by irritation of the motor pontile region; irritation of the fillet or tegmental area in the pons does not cause general epileptic convulsions. The pontile ganglion cells are endowed with epileptogenic properties, and convulsions from irritation of the pons are produced by means of these cells. The discharge of the pontile ganglion cells is conveyed to the cerebellar hemispheres by means of the middle cerebellar peduncles, and from the cerebellum to the spinal cord by means of the restiform bodies. The existence of subcortical epilepsy, as well as of cortical epilepsy, has been demonstrated in man. In genuine epilepsy the pontile gray matter is probably involved secondarily. The pontile convulsions are characterized clinically by less intensity of spasm, and by more involvement of the muscles of the trunk than of those of the limbs.

SPILLER.

7. UN CAS DE MERALGIE PARESTHÉSIQUE TRAITÉ PAR LE RÉSECTION DU NERF FEMORO-CUTANÉ (A Case of Meralgia Paresthetica Treated by the Re-section of the Femoro-cutaneous Nerve). A. Souques (*Revue Neurologique*, July 30, 1899, No. 14).

A young woman (saleswoman) of 21 years, normal in every way, found that she became very fatigued after a day's work. She was compelled to work twelve hours a day, during a large part of which she had to stand up. This occupation was followed for six years. She was compelled to change her employment, but with no special benefit. After a day of much fatigue, she felt in the left thigh at the level of the trochanter creeping and pricking sensations. These disappeared in a reclining position. Fifteen days afterward a sudden and severe pain attacked her in the same region, which prevented her from walking. This pain increased so much that morphine was required. These attacks of pain disappeared completely at times, especially when the

patient could rest during her vacation, but they soon reappeared more severe than ever. The painful area became larger and a burning sensation was added. The duration of an attack was from ten to fifteen minutes. Walking was impossible. In the absence of the paroxysms, the skin in the trochanteric region felt dead and cold. This condition resisted the usual therapeutic measures; electricity, cautery, methyl chloride, salicylate of methyl, etc.

On examination an area of the size of the palm of the hand, anesthetic to all sensory impressions, was found anteriorly over the left trochanteric region. This area was bounded by a region of very well marked hyperesthesia, which extended anteriorly to the median line of the anterior surface of the thigh, posteriorly four fingers' breadth, from the fold of the buttock, in height to the iliac crest and depth to the middle of the thigh. On account of the severity of the pain, and its interference with an active life, an operation was decided upon. A resection of the nerve at the level and above the crural arch was done; about three to four cm. were resected. After the operation, there was a total disappearance of the pain, but the area supplied by the nerve remained anesthetic. General improvement of the patient was noted. A sufficient time has not elapsed to observe the permanent value of the operation.

The pathology of this case is probably a compression of the femoro-cutaneous nerve, sometimes at the crural arch, sometimes at the level of the canal of the fascia lata. The author believes that in cases of meralgia paresthetica of traumatic origin, with symptoms as severe as in this case, an operation is justified.

SCHWAB.

#### ANATOMY AND PHYSIOLOGY.\*

8. UEBER DIE MOTORISCHEN LEITUNGSBAHNEN UND DIE ENTSTEHUNGSWEISE EPILEPTISCHER ANFÄLLE (Concerning the Motor Tracts and the Origin of Epileptic Attacks). Ernst Bischoff (Wiener klin. Wochenschrift, No. 39, Sept. 28, 1899, p. 961).

The experiments conducted by Bischoff are important, as they throw some light upon the causation of epileptic convulsions. He believes that the cerebellum probably has no part in the transmission of cortical impulses to the peripheral motor nerves, as, after extensive lesions of the cerebellum, even after extirpation, cortical irritation caused movements. A weak faradic current, which caused no contractions when applied to the cortex, produced strong tonic contractions of the opposite extremities when applied over an anterior pyramid. If the pyramid were cut below the point of irritation the contractions ceased, but were renewed with increased force when the current was applied to the pyramid below the point of division. This proves that in the dog the pyramidal tract certainly has motor functions, but irritation of the tract causes only tonic contractions of the muscles of the opposite extremities. Irritation of the pyramids does not produce clonic contractions; the tonic contractions do not persist after the irritation ceases, and muscles other than those first affected do not become involved, even though the irritation is prolonged. Irritation of the tegmentum at the level of the lower olives produces somewhat different results; only tonic contractions of the opposite limbs are caused by such irritation, but the increased tonicity persists a little time after the irritation has ceased, and the extremities of the side to which the irritation is applied are involved if the irritation is prolonged. This would seem to show that motor tracts are in the tegmentum. The contractions from cortical irritation are the same after the pyramids are cut as when they are intact. When one pyramid was cut the irritation of the cortex on the same side as the lesion had to be greater, in order to produce contractions in the contralateral side of the

body, than was necessary for the production of contractions from the opposite hemisphere through the sound pyramid. When both tegmentum and pyramid on one side were cut, irritation of the cortex on the same side did not cause epileptic convulsions.

Bischoff's statements are:

1. An interruption of the pyramidal tract in the dog causes difficulty in the production of contractions in the muscles of the contralateral limbs from faradic irritation applied to the motor cortex corresponding to the tract.

2. Irritation of the cortical centers, however, causes isolated contractions in the contralateral extremities.

3. In spite of division of one or both pyramids, prolonged irritation of the motor cortex causes true epileptic convulsions.

4. It is uncertain whether the pyramidal tract in the dog and cat conducts the impulses necessary for epileptic convulsions. It seems improbable that it does, because after unilateral lesion of the tegmentum epileptic convulsions could not be produced in the contralateral extremities, and could not be produced when the lesion of the tegmentum was bilateral, notwithstanding that the pyramids were intact (in one case).

5. The pyramidal tract in the dog certainly has motor functions, because faradic irritation of the tract causes tonic contractions of the muscles of the contralateral extremities. A connection of the pyramid with the muscles of the homolateral extremities in the dog could not be demonstrated by direct faradic irritation.

6. Isolated lesion of the tegmentum seems to have no effect on the production of single contractions in the extremities of the side opposite to the irritated cortex. After extensive bilateral lesion of the tegmentum, when the pyramids were intact, single contractions were produced by a feeble current.

7. If the tegmentum and pyramid on one side are injured, irritation of the homolateral hemisphere causes more feeble contractions in the contralateral extremities than are seen in the homolateral extremities from irritation of the contralateral hemisphere.

8. After the thalamus, the subthalamie region and the pyramidal tract of one side have been destroyed, faradic irritation of the motor cerebral cortex on the same side produces no results. Such a lesion seems to destroy the connection between the hemisphere and the contralateral extremities.

9. Extensive bilateral lesion of the tegmentum and pyramid at the level of the pons seems to destroy completely the motor conduction, so that cortical irritation of either side is without any effect. Animals with such a lesion have persistent paralysis.

10. Contractions in the homolateral extremities from cortical irritation were seldom seen.

11. Division of one side of the tegmentum with partial or complete lesion of the homolateral or both pyramids at any part between the thalamus and the proximal end of the vagus nucleus prevents the production of epileptic convulsions in the contralateral extremities from faradic irritation of the cerebral cortex of the operated side.

12. Continued irritation of the cerebral motor cortex of the operated side caused an epileptic attack in the homolateral extremities following tonic contractions of the contralateral extremities.

13. The cerebral cortex may be intact, and may have a partial connection with the spinal cord, but epileptic contractions do not occur in the limbs contralateral to the injured pyramid. This seems to show that in the tegmentum some change occurs in the impulse necessary for the epileptic convulsions.

SPILLER.

9. ANATOMISCHE UND VERGLEICHENDE ANATOMISCHE UNTERSUCHUNGEN ÜBER DIE VERBINDUNG DER SENSORISCHEN HIRN-NERVEN MIT DEM KLEINHIRN, DIRECTE SENSORISCHE KLEINHIRNBAHN, etc. (Anatomical and Comparative-anatomical Investigation on the Connection of the Sensory Cranial Nerves with the Cerebellum. Direct Sensory Cerebellar Tract, etc.). L. Edinger (Neurologisches Centralblatt, Oct. 15, 1899).

In a previous study on human embryos and fish brains, L. Edinger concluded that the sensory fibers which came from the cerebellum and joined the sensory cranial nerves were derived from the "direct sensory cerebellar tract." At that time he showed that these direct sensory fibers were part of the acusticus, trigeminus and in all probability the vagus. As Edinger was unable to prove this by degenerative experiments, he himself was doubtful about its correctness. One fact, however, was proven. The fibers which came down in the neighborhood of the trigeminus root were derived from the cerebellum. At least they could be followed to the nucleus. In the fourth edition of his well-known work on "Nervöse Central Organe"—the term "Direct Sensory Cerebellar Tract" was changed to "Tractus Nucleo-Cerebellaris." The uncertain state of knowledge in regard to the sensory cerebellar tract exists in the text books at present. Edinger adds in this study some new facts upon both questions.

It was found that the sensory cranial nerves are connected with the cerebellum in two ways, one direct, the other through the primary terminal nucleus. The tractus nucleo-cerebellaris and the direct sensory cerebellar tract exist side by side.

1. Tractus nucleo-cerebellaris of the acusticus, glossopharyngeus and vagus can be actually demonstrated. This was proven by the examination of a brain, in which there existed an anomaly of development; in the complete absence of the right cerebellar hemisphere, and the right worm, of which only a small part remained. In this specimen the rich network of myelin nerve fibers, which always stream into the vagus nucleus laterally, was completely absent, on the side upon which the cerebellum was lacking.

The whole fiber network on this side had disappeared. This same condition existed in the dorsal acusticus nucleus on the floor of the fourth ventricle. Also in the nucleus of the glossopharyngeus and in the nucleus funiculi teretis.

2. The direct sensory cerebellar tract was demonstrated by the following observation:

The labyrinth of a dog was taken out. Many fibers were degenerated as far as the neighborhood of the ventricular worm. They terminated in the nucleus of Bechterew and its vicinity, especially in the dorso-lateral region. The existence of this tract in mammals can only be demonstrated in the acusticus.

The facialis of selachians is practically a sensory nerve. The facialis, acusticus, several vagi roots and trigeminus, were cut. Degenerated fibers could be followed directly to the cerebellum.

The relation then of the cerebellum to the sensory nerves may be stated as follows:

First. There is a direct cerebellar tract, the fibers of which originate from the ganglia and enter the root itself, into the cerebellum.

Second. There is an indirect relation, the tractus nucleo-cerebellaris. Its fibers originate from the region of the root nuclei and in the nuclei of all sensory cranial nerves, certainly in that of the vagus, glossopharyngeus and acusticus, probably also in the terminal nuclei of the fifth nerve. This has been anatomically, not degeneratively, shown.

SCHWAB.

## Book Reviews.

I SOGNI: STUDI PSICOLOGICI E CLINICI DI UN ALIENISTA (con 3 figure e 1 Tavola). Di Sante de Sanctis. Docente nella R. Università di Roma. Torino. Fratelli Bocca, 1899.

The study of man's sleeping moments has always had a peculiar charm that does not attach itself to his waking activities; for in them, at times it has seemed that the proprieties of the social structure have been laid aside and the more natural psychical man is revealed, in certain aspects at least. To the labors of many authors who have contributed anecdotes, hypotheses and experimental researches, de Sanctis adds the results of his investigations, and has caught up the many threads of other men's ideas, and has worked them into this interesting volume of 390 pages.

There are thirteen chapters; at the end of each of which there is given an excellent bibliography of the subject matter pertaining to the chapter. In all there are some 313 bibliographical references. This of itself in an index of the author's wide reading. We can but mention the titles of the separate chapters: Dreams and Mysticism, Ancient and Modern: Methods of Study of Dreams: Dreams Among Animals: The Dreams of Babies and Children; Dreams of the Aged: Dreams of the Adult: Dreams of the Neuropathic; this chapter treats of dreams among hysterics, epileptics and neurasthenics: Dreams of the Insane, including those affected with hallucinations, frenasthenia, paranoia and alcoholism: Dreams of Delinquents: Dreams and the Emotions: On Dream States and Psychoses: Psychophysiology of Dreams, including a study of experimental dreams and hypnotic dreamy states, and finally: The Marvelous in Dreams.

While the work is largely a compilation of the views of many writers, it is by no means lacking in original insight or philosophic thought. We would like to see it in English dress that it might have a wider circle of readers, as we believe it deserves such recognition.

JELLIFFE.

ANIMAL INTELLIGENCE: AN EXPERIMENTAL STUDY OF THE ASSOCIATIVE PROCESSES IN ANIMALS. Edward L. Thorndike, A.M. Monograph Supplement, No. 8. Psychological Review, June, 1898, pp. 109.

In this two-year research with cats, dogs, and chickens, many interesting and some important things are well set forth. Dr. Thorndike's monograph is a good example of a properly conducted scientific investigation, and it is written in a style quite characteristic of its vigorous author. Its results are suggestive of an explanation of phenomena to be traced in the human mind as well as in that of the brutes.

In general, the mode of the experiments was to induce the animals by their desire for food to get at this by working various mechanically contrived doors leading from the boxes in which they were kept. All data concerning these actions as regards distances, times, identification, etc., were carefully recorded, thus giving the experiments, as far as they go, a precision of result which most observations on animal habits, etc., entirely lack.

The first result of the work is an accurate idea of just what things a cat or dog or chick can learn, what things are easy, and just how long each association is in forming. The next result is a final disproof of the theory that the acts of animals involve reasoning, comparison,



inference. The disproof consists in the fact that my animals did do by chance, and learn by association, representative acts (*e. g.*, using thumb-latch and button) of those which have been fancied to require reason. Further they would, in the case of some difficult associations, happen to do the thing six or seven times, but after long periods of promiscuous scrabbling, and then forever after would fail to do it. If they had acted from inference in any case they ought not to have failed in the seventh or eighth trial. What had been inferred six times should have been inferred the seventh. Finally, in all associations, the decrease in the time taken is gradual. Even after doing the thing the animal does not know enough to realize what it has done, and thereafter do it as soon as put in the box."

This opinion is wholly negative, as regards imitation in cats, dogs and chickens, the conclusion being that "no association leading to an act could be formed unless there was included in the association an impulse of the animal's own." He quite excludes any notion of idea, therefore, from the animals on which he worked. The fundamental thing in animals is impulse, such, *e. g.*, as a man has as his active consciousness when he plays an athletic game. The memory of these animals is considered purely neural and motor.

Suggestions for educational science, anthropology, and psychology arising from these considerations are appended, and form an interesting part of the work. Eleven pages of statistical time-curves and several descriptive illustrations doubtfully elucidate the text.

If the principles of this researcher's conclusions were to be disproven it could be done only by work yet more patient and "scientific" than that of Dr. Thorndike here described, and this were a difficult task. It seems to stand to-day really the last, and in general the best, work on a very essential problem.

DEARBORN.

ESSENTIALS OF DISEASES OF THE SKIN, INCLUDING THE SYPHILODERMATA. Arranged in the form of questions and answers. Prepared especially for students of medicine by Henry W. Stelwagon, M.D., Ph.D., Clinical Professor of Dermatology in the Jefferson Medical College; Physician to the Department for Skin Diseases, Howard Hospital; Dermatologist to the Philadelphia Hospital, etc. Fourth edition, thoroughly revised. Illustrated. Philadelphia: W. B. Saunders, 925 Walnut street. 1899.

The fourth edition of this compend does not markedly differ from its predecessor. Dermatology has not made such rapid advances in the last few years as to greatly affect the literature, and but few changes have been necessary to make this little volume representative of the science as it is taught to-day. In addition to these, descriptions have been added of some of the rarer affections not previously mentioned. The general excellence of the various members of this series of students' quiz books is so widely recognized that further particularization of the merits of this one is hardly necessary; it is sufficient to say that in all respects it is quite up to the high standard of its fellows and may safely be recommended to the student.

VOGEL.

HANDBUCH DER ANATOMIE UND VERGLEICHENDEN ANATOMIE DES CENTRALNERVENSYSTEMS DER SAUGTHIERE. Von Dr. Edw. Flatau und Dr. L. Jacobsohn. Bd. I. Makroskopischer Teil. Verlag von S. Karger, Berlin, 1899.

It would be practically impossible in a short review to even touch upon the many estimable features of a work so extensive as the one before us. Workers in experimental medicine have long since felt the want of a treatise on the comparative anatomy of the nervous system.

Apart from the few remarks in Edinger's and Kölliker's textbooks, medical and biological literature are practically barren of any extensive work on this subject. Under such conditions a treatise of any kind would have been acceptable; but a work so comprehensive in its scope, so thorough in its elaboration, and so finished in its pictured and written descriptions not only fills a long-felt want, but fills it so thoroughly and completely as to leave little cause for complaint in the future. This volume is devoted entirely to a consideration of the gross appearances. The relations of the different portions of the encephalon to each other and to corresponding portions in the brains of other types, and the anatomical relations of the convolutions to the superficial structures are considered under appropriate headings.

In the arrangement of the book, the higher types are considered first, and after these the next lower types are taken up seriatim. To those already conversant with the detailed anatomy of the human brain, this arrangement is probably the best that could have been selected. For the student of biology, or of veterinary medicine, however, an arrangement just the reverse of this would be better suited for the elucidation of the subject. The book, however, is intended for the investigator and not for the student; it is a practical work for facilitating the study of the nervous system by work on the lower animals, although the scientific side of the subject, such as would appeal to the biologist, has not been slighted. In many of the chapters the brain of one member of a family is taken as a type and described, and the other members considered in their relation to the type described. The simiæ are represented by the chimpanzee, orang-outang, macacus and hapale. The second chapter is devoted to the prosimii. Under this heading maki and stenops are studied in detail, and several other genera receive brief consideration. The third chapter is taken up with the very important consideration of the field mouse. Among the carnivora, the dog, cat and badger are described in detail. Chapter seven is devoted to a very careful, detailed discussion of the brain of the rabbit, guinea pig and rat. The remaining chapters of the book deal with the pig, horse, elephant, squirrel, manatee, porpoise, dolphin, echidna, duck bill, etc.

The tables devoted to the elaboration of the structural relations between the convolutions and the cranial bones show much painstaking labor and add considerably to the value of the work. The same might be said of the drawings in the text devoted to the same object. The extensive tables devoted to a comparison of absolute and relative brain weights are also evidence of the care taken in the preparation of the work. The determination of the relative and absolute brain weight is not of such practical importance as its place in the book would seem to indicate.

The book has 126 illustrations in the text and twenty-two illustrations on seven plates.

If there be room for any criticism of the work it is that in the description, very often the brain of an individual is taken as a type, and the variations in the arrangement in the convolutions of different individuals are left out of consideration. Such minor anomalies of arrangement which occur so frequently as almost to come under the pale of normal structure, often puzzle and at times mislead the operator on the cortex of the lower animals. Too much cannot be said in praise of the work, and no investigator in the field of experimental medicine, especially in that pertaining to neurology, can afford to be without it.

MCCARTHY.

XIIITH INTERNATIONAL MEDICAL CONGRESS,  
PARIS, 2-9 AUGUST, 1900.

AMERICAN NATIONAL COMMITTEE.

DR. HENRY BARTON JACOBS, Secretary, 3 W. Franklin St.,  
Baltimore, Md.

The American National Committee of the Thirteenth International Medical Congress is to be held in Paris from the 2d to 9th of August, 1900, in connection with the French Exposition.

All doctors of medicine are entitled to membership in this Congress by making the proper application and paying the sum of \$5.00. The Secretary-General in Paris has instructed the American National Committee to receive the applications of American physicians, and for this purpose a blank form is sent to each applicant, upon which is to be written full name and address, degrees and any position of note held, together with the Section of the Congress to which the writer wishes to belong. A visiting card should also be appended. These forms, with the \$5.00, are to be returned to the Secretary of the National Committee, Dr. Henry Barton Jacobs, 3 West Franklin street, Baltimore. He in turn will send receipt and forward the slips and money to Paris, where they will be registered, and in due course of time a card of admission to the Congress mailed to each applicant.

Members desiring to present papers will forward the title and an abstract of the paper before May 1, 1900, to the Secretary of the Section to which they belong, for each Sectional Committee reserves to itself the right of drawing its own working programme. Papers are limited to fifteen minutes.

The committee hopes the American representation in this extremely important Medical Congress may be as large as possible, and they would urge every member of the profession to enter his name for membership, this alone entitling him to receive a digest of the full proceedings of the Congress and the printed report of the section to which he belongs. Communications respecting the delivery of these reports to members should be addressed to M. Masson, publisher of the proceedings of the Congress, 120 boulevard St. Germain, Paris.

SECTION OF NEUROLOGY.

President, Raymond; vice-presidents, Brissaud, Dejerine, Grasset (Montpellier); Pitres (Bordeaux); secretary, P. Marie, 3 rue Cambacérés, Paris; members, André (Toulouse); d'Astros (Marseilles); Babinski, G. Ballet, Bernheim (Nancy); Binet, Bourneville, Jean Charcot, Ch. Féré, Gilles de la Tourette, A. Gombault; Haushalter (Nancy); Hayem, Klippel, Launois; Mirallié (Nantes); Parisot (Nancy); Pierret (Lyons)); Rauzier (Montpellier); Paul Richer.

PAPERS.

1. On the Centers of Projection and of Association in the Human Brain, by Flechsig (Leipzig); Hitzig (Halle); von Monakow (Zurich).
2. On the Nature and Canalisation of Tendinous Reflexes, by Jendrassik (Budapest); C. S. Sherrington (Liverpool).
3. Nature and Treatment of Acute Myelitis, by von Leyden (Berlin); Marinesco (Bukarest); Crocq (Bruxelles).
4. Diagnosis of Organic Hemiplegia with Hysteric Hemiplegia, by Roth (Moscow); Ferrier (London).
5. On Non-tabetic Lesions of the Posterior Branches of the Medulla, by Bruce (Edinburg); Hömen (Helsingfors).
6. On Various Points in the Study of Aphasia, Tamburini (Reggio); Pick (Prague).

## SECTION OF PSYCHIATRY.

President, Magnan; vice-presidents, Joffroy, Gilbert Ballet; Pierret (Lyons), and Culerre (Laroche-sur-Yon); secretary, Ant. Ritti (Asile de Charenton, Seine France); members, Bouchereau, Bourneville; Albert Carrier (Lyons); Christian (Charenton); Doutrebente (Blois); Jules Falret, Ch. Féré; Febvre (Ville-Evrard); P. Garnier, Giraud (Saint-Yon); Mairét (Montpellier); Meuriot, Motet; Parant (Toulouse); Régis (Bordeaux); Seglas; Taguet (Vaucluse); Vallon, Jules Voisin.

## PAPERS.

1. Mental Pathology—Psychoses of Puberty, by Ziehen (Jena); Marro (Turin); J. Voisin (Paris). 2. Pathological Anatomy—Pathological Anatomy of Idiocy, by G. E. Shuttleworth and Fletcher Beach (London); Mierzejewski (St. Petersburg); Bourneville (Paris). 3. Therapeutics—On the Confinement to Bed in the Treatment of Acute Forms of Insanity and on the Modifications that it Might Entail in the Establishment of Houses for the Insane, by Clemens Neisser (Leubus); Kosakof (Moscow); Morel (Mons). 4. Legal Medicine—Sexual Perversion with Obsession and the Impulses from the Medico-Legal Point of View, by von Krafft-Ebing (Vienna); Morselli (Genoa); Paul Garnier (Paris).

## BOOKS RECEIVED.

"Nervenleiden und Erziehung," von Prof. H. Oppenheim. Berlin, S. Karger.

"Die Erkrankungen des Nervensystems nach Unfällen, mit besondere Berücksichtigung der Untersuchung und Begutachtung," von Dr. Heinrich Sachs und Dr. C. S. Freund. Berlin, Fischus-Medicinische Buchhandlung, H. Kornfeld.

"Travaux du Laboratoire de Neurologie." Pub. par A. Van Gehuchten, Université de Louvain Institute Vésale.

"Clinical Lectures on Neurasthenia," by Thomas D. Savill, M.D., London. Wm. Wood & Co., N. Y.

"Text-Book of Embryology," by John C. Heisler, M.D. W. B. Saunders, Phila., Pa. Illustrated.

"Lectures upon the Principles of Surgery," by Chas. B. Nancrede, M.D., with an appendix by Wm. A. Spitzley, M.D. W. B. Saunders, Phila. Illustrated.

"Essentials of Physical Diagnosis of the Thorax," by A. M. Corwin, M.D. W. B. Saunders, Phila.

"Essentials of Anatomy," by Chas. B. Nancrede, M.D. W. B. Saunders, Phila.

"Essentials of Diseases of the Skin," by Henry W. Stelwagon, M.D. W. B. Saunders, Phila. Illustrated.

"American Text-Book of Surgery," W. W. Keen, M.D., and J. Wm. White, M.D. W. B. Saunders, Phila.

"Practice of Medicine," James M. Anders. W. B. Saunders, Phila. Illustrated.

"Text-Book upon the Pathogenic Bacteria," by Jas. McFarland, M.D. W. B. Saunders, Phila. Illustrated.

"Text-Book of Materia Medica," by Geo. F. Butler, M.D. W. B. Saunders, Phila.

Transactions of the Iowa State Medical Society. Vol. xvii. Forty-Eight Annual Session, 1899. H. G. Middleditch, Waterloo, Iowa.

Progressive Medicine, Vol. iv., December, 1899. H. A. Hare, M.D. Lea Bros. & Co., Phila.

Archives of Neurology & Psycho-Pathology, Vol. ii., Nos. 1 & 2, 1899.

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THE  
**Journal**  
OF  
**Nervous and Mental Disease.**

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**Original Articles.**

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A STUDY OF THE LESIONS IN A SECOND CASE OF  
TRAUMA OF THE CERVICAL REGION OF THE SPINAL  
CORD, SIMULATING SYRINGOMYELIA.\*

BY JAMES HENDRIE LLOYD, A.M., M.D.,  
NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

In this JOURNAL in June, 1894, I reported two cases of traumatic affections of the cervical region of the spinal cord simulating syringomyelia. Both these cases occurred in men who had sustained severe injuries, involving fracture of the cervical spine, many years before. In both cases the resemblance to syringomyelia was marked. There were atrophy and paralysis of the shoulder and arm muscles, with fibrillary contractions, and spastic paraplegia without atrophy in the leg muscles, and without involvement of the bladder and rectum. Both patients had the dissociation symptom well marked. There was preservation of tactile sense, with loss of sensation for heat, cold, and pain; and, what was most striking, this dissociation symptom was unilateral. In one case, however, the paralysis was hemiplegic in type, and the dissociation symptom was entirely confined to the side opposite the paralysis. Thus the Brown-Séquard syndrome, modified, however, by the absence of tactile anesthesia, was presented; and this rare phenomenon of a crossed hemiplegia with the dissociation symptom, as a result of trauma, was one of the earliest instances that had been reported in the literature of the subject. The patient in this lat-

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\*Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

ter case having subsequently died, a full report of the autopsy was published in the spring number of *Brain*, 1898. In that paper it was shown that the lesion was confined largely to one side of the cord, and certain inferences were drawn from the findings as to the probable course of the various sensory fibers in the cord. The hemiplegia had existed on the side of the lesion; due, of course, to involvement of the lateral tract; while the involvement of sensation for heat, cold and pain on the side opposite the lesion was explained by the probable decussation of the fibers for these modes of sensation in the gray matter, and their subsequent course cephalad by way of Gowers' tract. The preservation of tactile sense was explained by the fact that the posterior columns were but little involved in the lesion. The case was a particularly favorable one for such a study, because the lesion was practically unilateral and involved almost the entire half of the cord, the part that had been the least injured being the posterior column.

I have now the opportunity to report the results of the autopsy in the other of these two cases. Before doing so, however, I shall present a brief abstract of the clinical report from my preceding paper (*op. cit.*).

James G., aged 55, a stone-cutter by trade, of good family and personal history, had met with two severe accidents. In 1876 he fell from a scaffolding and sustained, apparently, a fracture of the cervical spine. According to his own report his entire body was paralyzed, breathing was embarrassed, and urination, but not defecation, was affected. He stated that there was loss of sensation in the arms, but none below the waist; but probably not much reliance can be placed on this report of the sensory involvement. The essential facts are that he slowly recovered power—in the arms more slowly than in the legs—and was able at the end of two years to return to his work as a stone mason. His neck was deformed in a way that plainly indicated that the man had had a fracture of one or more vertebræ. The fact that he was able to return to work at all, and especially to such heavy work, is remarkable. That the cord had suffered injury in this first accident seems indubitable, and that this injury would have laid the foundations, in part at least, for subsequent degenerative changes, is probable, even if the man had not suffered a second accident. Five years after resuming work (*i. e.* in 1884) the patient had a second fall, this time from a ladder, a distance of fifteen feet,

striking the back of his head and the spine between the shoulders. He was unconscious for six hours. This fall was followed by a return of the paralysis of the arms and legs, loss of control over the bladder, and obstinate constipation. According to the patient's own statement there was no marked loss of sensation, but we do not know how carefully that was determined. There was no priapism. There was severe pain at the seat of injury, and headache for three months. Power



Fig. I.—Trauma of the cervical region of the spinal cord.

returned in the legs in two months, in the arms slightly by the end of seven months. After confinement to bed for four months the patient began to get about, but he was never able to work again after the second accident.

In 1894 the patient came under my care in the Philadelphia Hospital and continued there until his death in 1899. His condition when first seen was, in brief, as follows: There was advanced atrophy of the shoulder muscles of both sides, including the deltoid, pectoralis major, infra and supraspinati

and the lower part of the trapezius; also the muscles of arms, forearms, and of almost all the muscles of the hands. Fibrillary contractions were active in some of the wasted muscles. The myotatic irritability was much increased. The arms were completely paralyzed, except for a slight power of flexion and extension of the forearm. The muscles responded to both

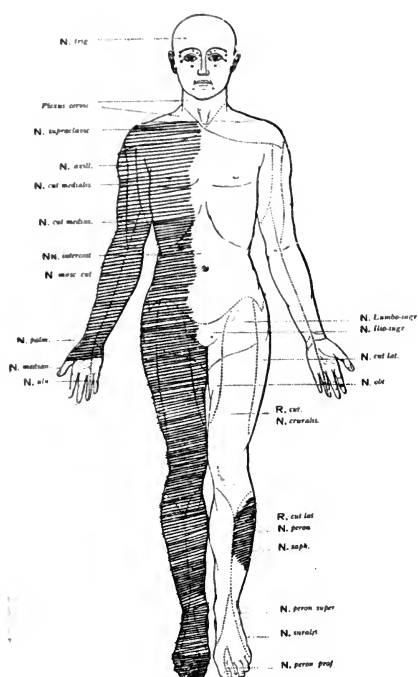


Fig. II.

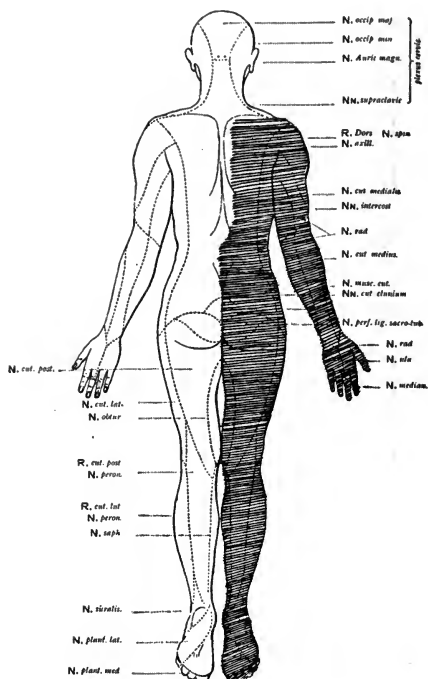


Fig. III.

Fig. II. Thermo-anesthesia and analgesia on right side. Tactile anesthesia in small area on left leg.

Fig. III. Thermo-anesthesia and analgesia on left side. Tactile anesthesia in small area on left leg.

currents; but there was decrease in excitability in the most wasted muscles, while there was hyperexcitability with duration tetany in others. There was no head-drop. The legs were paretic and contracted. The knee-jerks were exaggerated, and ankle-clonus was present. There was no muscular atrophy in the legs, and no trophic lesions, no bulbar symptoms, nor paralysis of the bladder. There was great deform-



ity of the cervical spine (see Fig. 1). The sensory symptoms were of great interest. There was still more or less neuralgic pain about the neck and occiput. Tactile sensation was good everywhere, as demonstrated by repeated tests. Thermo-anesthesia, or, more properly, anesthesia to cold, was marked on the right side as shown in the schemata. The patient distinguished heat better than cold, always calling cold hot. Analgesia was quite marked in some of this affected area, but was not coextensive with the anesthesia to cold. This condition remained unchanged for more than a year, during which time repeated observations were made.

The patient died in 1899, five years after the report of the case. During that time the symptoms remained practically unchanged. Death resulted from gradual exhaustion. Towards the end respiration became embarrassed, and this contributed directly to the death.

At the autopsy the cervical vertebræ, from about the 4th to the 7th were found much deformed and ankylosed, as the result of an old fracture. It was difficult to determine exactly what had been the extent of the injury, because of the state of ankylosis with formation of callus. As the chief interest of the case centers upon the injury to the spinal cord, I shall simply report the extent and appearances of the lesion in the spinal medullary tissue. There was some thickening of the dura mater overlying the seat of fracture, but it was hardly sufficient to be an important factor in the causation of the symptoms. The cord underlying the fracture was deformed and somewhat flattened. It was evidently extensively degenerated and sclerosed.

The microscopic appearances are as follows: At the point of greatest injury the cord is deformed and extensively degenerated. The gray matter is especially involved. It is difficult to distinguish clearly even the outlines of the anterior horns, and the multipolar cells seem to have almost disappeared. The posterior horns are more clearly discernible. The white matter is also widely degenerated. This is so especially of the antero-lateral columns; consequently the lateral pyramidal tracts, the direct cerebellar tracts and the antero-lateral ground bundles, with Gowers' tracts, all on both sides, are deeply involved. The only parts of the cord that seem to have maintained their integrity to any extent are the posterior columns, and to some extent the direct pyramidal tracts. This destruction of the gray matter and approximate preservation of the posterior columns are of great significance in view of the clinical phenomena presented by the case.

Above the seat of lesion the degeneration of the direct cere-

bellar tracts can be traced into the medulla. There is no degeneration of the fillet, nor of the pyramids.

Below the seat of lesion there is the characteristic descending degeneration of the lateral tracts. It presents nothing worthy of special note. The degeneration is complete as far as the tract can be followed into the lumbar enlargement, where it approaches the periphery as it diminishes in size. A point of special interest is the evidence of some slight and limited degeneration in the posterior columns below the cervical lesion. This is best marked in the cervical cord itself just below the seat of injury. The parts most sclerosed are the columns of Goll. The sclerosis exists as a wedge-shaped area, very similar to what is seen in ascending degeneration of Goll's columns in case of a transverse lesion in the lower dorsal or lumbar cord. As there is no lesion in this cord below the cervical region, this degeneration in Goll's columns cannot be properly an ascending one. It may possibly represent the degeneration of the descending sensory fibers after the dichotomous division of the entering neuraxon of the sensory neuron-body located in the posterior ganglion. Why it should so distinctly occupy Goll's columns to the exemption of the rest of the posterior columns is not apparent. The important fact remains, however, that even at the seat of greatest injury the posterior columns are the least injured of any portion of the cord, and this fact evidently has important relation with the clinical fact that the patient did not have impaired tactile sensibility. This slight degeneration in Goll's columns can be traced in diminishing intensity and extent as far as the dorsal region, where it is lost—a fact which argues against it being a true ascending degeneration. In the lumbar region the posterior columns—both Goll's and Burdach's—are absolutely intact. There is evidence in this cord of degeneration in the comma zones of Schultze.

In attempting to correlate the clinical phenomena in this case with the anatomical findings, I regard the following facts as conspicuous: There is extensive destruction of the gray matter and of the direct cerebellar tracts and Gowers' tracts, and comparative exemption of the posterior columns. This exemption of the posterior columns is, to be sure, not complete, but it is probably sufficient to explain satisfactorily the preservation of tactile sensibility. The inference, therefore, seems warranted, just as in the former case, that the tracts for heat, cold and pain pass up by way of the gray matter, eventually passing from the gray matter into Gowers' tracts; while

tactile, or common, sensation is conveyed by the fibers in the posterior columns. Thus would be explained the analgesia and thermo-anesthesia—*i. e.* as due to the destruction of the gray matter; while the preservation of tactile sensibility is explained by the comparative integrity of the posterior columns. There is no cavity formation anywhere in this cord, but the seat of the lesion in the gray matter is the same practically as in most cases of syringomyelia.<sup>1</sup> In drawing this deduction I am in accord, in part at least, with some of the most competent observers. Van Gehuchten has summed up our knowledge on these points as follows: The fibers of the posterior columns, derived from the neurons in the posterior ganglia, serve for the conduction of tactile sensibility; these fibers are direct, *i. e.*, not crossed, and terminate in the nuclei of the posterior columns; in other words, the nuclei graciles and nuclei cuneati. On the other hand, the impressions of heat, cold and pain are transmitted by way of the gray matter, through a second order of neurons, the cell bodies of which are located in the posterior horns and the neuraxons of which cross to the opposite side and pass cephalad by way of Gowers' tract. Van Gehuchten refers to the lesion of syringomyelia as demonstrating this probable course of the various sensory tracts. In my first case of trauma of the cervical cord (reported in *Brain*) this arrangement was clearly indicated by the fact that the lesion was almost entirely unilateral, and the thermoanesthesia was on the side *opposite* the lesion, while the tactile anesthesia and hemiplegia were on the *same* side as the lesion. In this respect that case was probably unique in the literature of this subject. In the present case the lesion is not unilateral, although the thermoanesthesia was confined to one side—a condition which may be explained by the fact that the clinical study was made some years before the patient's death, and, therefore, progressive degeneration may in the meantime have involved practically the gray matter on both sides.

In a volume of clinical lectures recently published, Brissaud<sup>2</sup> has illustrated this subject from some cases of spinal syphilis.

<sup>1</sup> The microscopic sections were kindly made for me by Dr. David Riesman.

<sup>2</sup> "Leçons sur les mal. nerv." Deux. Série, Paris, 1899.

His explanation is practically the same as that given in the report of my case in *Brain*, in 1898. He especially emphasizes the possibility of the occurrence of the Brown-Séquard syndrome without loss of tactile sensibility—in other words, a hemiplegia with a crossed dissociation symptom. This, as already stated, was the exact condition in my first case. Brissaud, in order to illustrate his meaning, uses an ideal scheme, which almost exactly represents the real lesion in my case. Dejerine and Sottas<sup>3</sup> were among the first to record an instance of this crossed thermoanalgesia from a unilateral lesion, although the condition was not so striking as in my case, because there was thermoanalgesia of limited extent on the same side as the lesion also. The case was one of syringomyelia with a cavity almost limited to the right posterior horn; tactile sensibility was normal, but thermoanalgesia was present in the thorax and upper extremity on the side of the lesion and on the whole of the opposite side of the body. From the purely anatomical standpoint all such cases are in accord with the views of Edinger, who teaches that the sensory fibers which decussate in successive stages in the gray matter (the fibers here alluded to) do not decussate again in the medulla oblongata, but continue their course towards the cerebrum on the side opposite their entrance into the cord, and are rejoined by the fibers issuing from the nucleus gracilis and nucleus cuneatus—fibers which cross in the decussation of the lemniscus and constitute the arciform fibers.

In the latest (3d) edition of his book Van Gehuchten, summing up our knowledge of the course of the sensory fibers in the cord, concludes that the impressions of pain, heat and cold are transmitted by Gowers' tracts. As these tracts are made up largely of fibers arising from nerve cells in the gray matter on the opposite side, this conclusion is in accord with the pathological findings in my first case especially. In the present case the lesion in the gray matter is not sufficiently unilateral to permit this deduction, but the case at least proves that these modes of sensation are not conveyed by the posterior columns. Van Gehuchten inclines to adopt the re-

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<sup>3</sup> Bull. et Mém. de la Soc. de Biol., 25 Juil., 1891.

sults of Langendorff's experiments, which, he thinks, seem to prove that the posterior columns do not convey tactile impressions, but that these impressions must also pass through the gray matter, possibly reaching the brain by way of the direct cerebellar tracts. But such a conclusion, contrary to all clinical experience, does not seem to me warranted by Langendorff's experiments, which consisted in ligating the abdominal aorta (thus, it is claimed, invalidating the gray matter) and then noting alterations in the blood pressure upon irritating the skin—surely a method which takes much for granted. It seems, on the contrary, that we are still warranted in regarding the posterior columns as the paths for tactile, or common, sensation.

On the clinical side, which Van Gehuchten himself acknowledges is the only safe one for determining the sensory functions of the gray matter, several recent cases have been put on record. Among these is the case reported by Cushing,<sup>4</sup> in which a pistol shot wound of the spinal cord caused the dissociation symptom most marked on the side opposite the lesion and opposite the paralysis. The patient recovered, so, of course, the lesion could not be determined. Van Gehuchten<sup>5</sup> has recently contributed a paper on the dissociation symptom caused by traumata of the spinal cord.

10. SUR LES ALTERATIONS DES GRANDES CELLULES PYRAMIDALES CONSECUTIVES AUX LESIONS DE LA CAPSULE INTERNE (Changes in the Large Pyramidal Cells Resulting from a Lesion of the Internal Capsule). Marinesco (Rev. Neurol., 7, 1899, p. 359).

Six cases of hemiplegia were examined by Marinesco in which the internal capsule had suffered injury, with special reference to the large pyramidal cells of the cortex in the paracentral, frontal, and parietal convolutions. He found that the changes consisted in atrophy and degeneration of these cells analogous to the changes in the cells of the anterior horn after lesions and alterations in the peripheral nerves. The large pyramidal cells were the only ones affected, while the other cells of the cortex remained intact. Investigation of lesions more deeply seated and further removed, *e. g.*, in the pons or cord, showed that the further away from the cortex the pyramidal tract is injured, the later and less intense are the changes in the large cortical cells ("reaction at a distance").

JELLIFFE.

<sup>4</sup> Am. Jour. Med. Sci., May, 1898.

<sup>5</sup> La Semaine Méd., 1899.

## LANDRY'S PARALYSIS.\*

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In a paper presented to this Association two years ago, one of us (Thomas) from a study of the anatomical changes in two cases of acute ascending paralysis and of the cases with autopsies reported in the literature of the subject, came to the conclusion that the anatomical basis of the disease was an acute parenchymatous degeneration involving the peripheral motor neurone, arising from some toxic or infectious cause. In a paper presented to the Association a year ago, Mills and Spiller<sup>2</sup> came independently to a similar conclusion. The results of the autopsy in a third case which we present to-day are confirmatory of the conclusion previously reached.

As the evidence in favor of this conclusion is now so strong it seems hardly necessary to review in detail the results of a considerable number of autopsies reported since these two papers were published. Krewer<sup>3</sup> in three cases found subacute neuritis with acute myelitis; Schultz,<sup>4</sup> in one case acute diffuse myelitis with chronic neuritis; Roger and Josué<sup>5</sup> found changes in the anterior horns and pneumococci; Piccinino,<sup>6</sup> changes in the gray matter of the cord with the diplococcus intracellularis; Worcester,<sup>7</sup> changes in the anterior horns, peripheral nerves and cerebral cortex; Hirtz and Lesné,<sup>8</sup> changes in the an-

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\*Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

<sup>1</sup> Thomas, Amer. Journ. of the Med. Sciences, August, 1898.

<sup>2</sup> Mills and Spiller, JOURN. OF NERVOUS AND MENTAL DISEASE, June, 1898.

<sup>3</sup> Krewer, Zeitschrift für klin. Med., xxxii, 115.

<sup>4</sup> Schultz, Norsk Mag. f. Lægevid, No. 6, 1898, abstract in Revue Neurologique, 15 Jan., 1899.

<sup>5</sup> Roger and Josué, Press méd., 27 July, 1898.

<sup>6</sup> Piccinino, Annali di Neurologia, xv, 1, 1897.

<sup>7</sup> Worcester, JOURNAL OF NERVOUS AND MENTAL DISEASE, May, 1898.

<sup>8</sup> Hirtz and Lesné, Presse méd., 12 June, 1897.

terior horns without any neuritis; Burghart,<sup>9</sup> in a case that had nearly recovered from the paralysis, changes in the anterior horns with very slight changes in the endoneurium; and Chantemesse and Ramond,<sup>10</sup> in two cases which perhaps should not be classed as Landry's paralysis, but as beri-beri, changes in both cord and nerves, with bacilli in the cerebrospinal fluid which produced similar symptoms in animals. In favor of the toxic-infectious origin, moreover, beside the evidence of the clinical cases, Remlinger<sup>11</sup> claims to have produced acute ascending paralysis in rabbits by inoculation with pathogenic cocci, and Rendu<sup>12</sup> has observed the symptoms developing after inoculation of anti-rabic serum.

Although the affection is probably of toxic-infectious origin, the evidence in favor of a bacterial origin, in spite of Remlinger's experiments and the cases reported by Roger and Josué, Chantemesse and Ramond, and Piccinino, as well as the cases previously reviewed by one of us, is not conclusive. In the majority of cases, including the two previously reported by one of us, and the case at present under consideration, the results of bacteriological examination have been negative.

In Landry's original case and in a considerable number of cases reported since, the anatomical changes were reported negative. Many of those cases, however, can not at present be cited as evidence, since they were examined before the present microscopical technique had been introduced. A few cases, however, reported recently demand a brief consideration. Schultz,<sup>13</sup> in an article accessible only in an abstract, speaks of a case of acute ascending paralysis in an alcoholic patient which proved fatal in fifteen days. Nothing was found in the nerves or cord, but no statement is made in the abstract as to the methods employed. Goebel,<sup>14</sup> in a case of seventeen days' duration, found the peripheral nerves normal (osmic acid, Weigert, carmine); in some muscles fresh degeneration of the muscle fibers and occasionally increase of interstitial tissue; in

<sup>9</sup> Burghart, *Charité Annalen*, xxii, 1897.

<sup>10</sup> Chantemesse and Ramond, *Revue Neurologique*, 30 Nov., 1898.

<sup>11</sup> Remlinger, *Revue Neurologique*, 15 April, 1898.

<sup>12</sup> Rendu, *Revue Neurologique*, 15 July, 1897.

<sup>13</sup> Schultz, *art. cit.*

<sup>14</sup> Goebel, *Neurologisches Centralblatt*, 1 April, 1898.

some bundles of the cauda equina focal destruction of nerve fibers in the neighborhood of a vessel distended with blood, with fresh degeneration (Marchi) of some axis cylinders and medullary sheaths; the spinal cord (Weigert, carmine, Marchi), and the anterior roots (Weigert, carmine, osmic acid) were normal. From the decussation of the pyramids to the oculomotor nuclei some slight degeneration in the long tracts and the nerve roots could be detected by Marchi's method. In the discussion Nonne reported a case with marked bulbar symptoms where both the medulla oblongata and the cranial nerves were normal. In both cases Nissl's methods gave no certain anomalies, but Nonne recalled the opinions of Goldscheider and Flatau that we must be cautious in estimating the value of changes found by these methods. It is again uncertain with the incomplete report how far we can regard "no certain anomalies" (keine sicheren Anomalieen) as absolute proof of negative findings. Girandeau and Lévi<sup>15</sup> have recently reported a case of ten days' duration, coming on during convalescence from a typhoid which had been treated with anti-typhoid serum, where after careful use of osmic acid, Pal's method, hematoxylin-eosine, picro-carmine, Nissl's method, neither they nor Nageotte were able to discover any changes.

This last case, at any rate, seems to indicate that even with modern technique no anatomical changes were found. To it, and to the others, must be opposed a much greater number of cases where modern methods show definite changes. Accepting these few cases as they stand, they simply show that in rare instances our modern methods are inadequate to reveal the changes produced by the toxic agent which gives rise to acute ascending paralysis. In the great majority of cases, however, our modern methods prove conclusively that there is an acute parenchymatous degeneration of the peripheral motor neurone manifesting itself most markedly at times in the ganglion cell, at other times in the axis cylinder process. Bodin,<sup>16</sup> has claimed that the whole nervous system from the

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<sup>15</sup> Girandeau and Lévi, *Revue neurologique*, 15 Oct., 1898.

<sup>16</sup> Bodin, "Essai sur les paralysies ascendantes aiguës." Thèse de Paris, 1896.



brain to the nerves may be attacked, but this is not confirmed either clinically or pathologically.

Krewer,<sup>17</sup> however, has advanced a theory, based upon four cases, of which three came to autopsy, which also derives some support from one of the cases already cited reported by Schultz. He found, as we have said, changes in the peripheral nerves indicative of chronic neuritis, together with changes in the cord indicative of acute myelitis. Of his fatal cases three were alcoholic and one also tuberculous, and all three had had influenza shortly before the onset of the acute paralysis. He maintains that Landry's paralysis is the second and third stages of a chronic multiple neuritis, which has extended to the spinal cord *per continuitatem*. In the latter it extends very rapidly, usually upwards, but sometimes downwards, and causes death by involvement of the vital centers in the floor of the fourth ventricle. A new factor in the form of an infectious disease is usually necessary to cause an outbreak of Landry's paralysis in an existing multiple neuritis. Neither in the two cases previously reported nor in the three we bring forward to-day was there any evidence of a pre-existing multiple neuritis. As a rule such a history is absent in the cases reported clinically by other observers, and in many cases with the autopsies the anatomical changes of chronic multiple neuritis have been absent.

Having spoken of the pathological basis of acute ascending paralysis we will now report three cases, one of which came to autopsy, one made a partial recovery, and the third recovered completely.

CASE I. On the fifth of March, 1898, we were asked by Dr. F. W. Anthony, of Haverhill, to whom we are indebted for notes in regard to the patient, to see Mrs. D., who had been ill for about a week.

She was born in Massachusetts, of American parentage, and was between twenty-seven and twenty-eight years of age. Her father had been a dissipated man; her mother had died of valvular disease of the heart, cirrhosis of the liver, ascites, and carcinomatosis. The patient herself had had, in childhood,

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<sup>17</sup> Krewer, *art. cit.*

measles, whooping cough, mumps and scarlet fever. Four years previously, she had an attack of influenza, when she slept for three days and nights, and had to be wakened to take food, immediately falling to sleep again. She had no physician at the time, and was up in about a week. She had always been subject to migraine up to about three years ago, since which time the attacks had been much less frequent. She had one child ten years ago, with normal labor; she had had no miscarriages. Her husband was a very dissipated man, and she had not lived with him for the past five years. She herself had indulged very slightly in alcohol, but never to excess, and it was reported that since leaving her husband she had lived with another man, but no history could be obtained of syphilis, and she had not led a dissipated life. She had had internal strabismus of the right eye since childhood, following a fall into a well. She had always been subject to constipation, and for the past year she had had a peculiar fluttering feeling in the back, but, on the whole, her health had been better for the last six months than it had ever been during her life.

On the 26th of February, when she arose in the morning, she noticed that the legs felt stiff and it was hard to get up. She could not go down stairs as well as usual, and after she went out she fell in stepping off the curbstone. On the 27th of February she fell while running across the floor, and subsequently during the day she fell repeatedly while walking, and the legs felt considerably weaker. On the 28th she was able to walk, but she fell more frequently. Dr. Anthony was called in, and he found that the power of abduction was lost in the left leg and the power of adduction was much diminished; abduction and adduction were diminished in the right leg; she was unable to flex the left leg at all, and she had great difficulty in flexing or extending the right leg upon the thigh; the knee-jerks were absent; there was no fever; no impairment of sensibility; no disturbance of the bladder or rectum; vaginal examination was negative.

March 1st. She was wholly unable to walk; the left leg was practically powerless; the right leg was very weak; she could not raise it in bed or abduct or adduct it at all; sensibility was normal; the arms were unaffected.

March 2d. The condition was much the same. There was tenderness on percussion on each side of the lumbar vertebræ.

March 4th. The grasp was diminished in both hands; the right pupil was somewhat dilated; she complained of some disturbance of vision, as if she saw objects through something like a veil; the pupils were normal and reacted to light; she

slept poorly and complained of inability to swallow solids and difficulty in swallowing liquids.

March 5th. We both saw her with Dr. Anthony, in consultation. She did not have much pain, but complained of some cramp in the abdomen and of some soreness in the head; the legs felt as if quivering or crawling, and she had some cramp or pain in them. There was a trembling, quivering feeling beneath the skin, in the legs and throat; she slept poorly, thinking constantly but not worrying; she had considerable palpitation and a beating feeling all over. There was no difficulty in talking, but fluids seemed to stick in the throat when she attempted to swallow. The appetite was good; the digestion unimpaired; the bowels and the bladder acted normally; there was no disturbance of the pelvic organs. She had never had any menstrual disturbances. Everything seemed blurred that she looked at, but there was no double vision. She was dressed and sitting in a chair; rather a slight woman, well developed, fairly well nourished and of good color. The right pupil was larger than the left; neither reacted to light or accommodation. Vision in the right eye had always been less than in the left. The visual field was normal; the ophthalmoscopic examination negative. The movements of the eyes, face, tongue and palate were normal. She could make all movements with the arms, but all the movements were weak. The movements of the ribs in respiration were equal, but not up to the normal; the movements of the abdominal muscles were fairly good; she could twitch the legs slightly at the trunk, but she could not flex the thighs upon the body or abduct or adduct the legs; there was a slight movement of rotation outwards; there was no motion at all at the knee-joint, and only a very slight plantar flexion of the feet. Sensation everywhere was good for touch, pain and temperature, but the right leg seemed somewhat hypersensitive to pain. There was considerable tenderness on pressure over the posterior tibial nerves; a slight tenderness on pressure over the nerves in the arm; there was also some tenderness over the vertebræ by the sides of the spine in the mid-dorsal region and over the sacro-iliac synchondrosis. The muscles of the lower leg did not react to a tolerably strong faradic current; the vastus internus reacted to a moderately strong current feebly, less upon the right side than upon the left; the muscles of the arms reacted normally to faradism. The knee-jerk was absent; the plantar reflex was absent on the right and much diminished on the left; the abdominal and epigastric reflexes were normal. Examination of the chest was negative. The splenic dullness extended between three and four centimeters on each side of the

costal border. The examination of the abdomen and of the pelvic organs was negative. The temperature was not elevated. A watch was heard at three inches from either ear; the bone conduction was equal; there was a slight weakness of the right levator palpebræ superioris.

March 6th. The grasp of both hands was much diminished, especially on the left side. She swallowed liquids with the greatest difficulty. She complained of headache, most severe in the occipital region. The abdominal muscles were not markedly affected. Sensation was apparently unimpaired. The right pupil was dilated and the vision was less distinct with the right eye. She was unable to move either leg. She was taken to the hospital.

March 7th. The tongue was protruded to the left. She had to be catheterized last night and this morning; she could take a small amount of nourishment by mouth. The feet and legs pained her a good deal during the night. She was given nutrient enemata every three hours.

March 8th. Some difficulty in articulation. Incontinence of urine twice during the night, but she passed urine naturally afterwards; later she had to be catheterized. She felt drowsy much of the time; the feet and legs pained her considerably during the night; the pupils were nearly equal, responding sluggishly to light. There was some tenderness on each side of the cheek, over the rami of the jaws and at the sides of the head in the parietal region.

March 9th. Vision remained about the same. The arms were in about the same condition. She felt drowsy; had a good deal of abdominal distress during the night; had a prickly sensation in the feet and arms during the night; she had a choking spell which exhausted her considerably.

March 10th. No incontinence during the night. She had great pain in the abdomen, relieved by  $\frac{1}{3}$  of a grain of morphine. She was considerably weaker and drowsy. She had some trouble with mucus in the throat, which she coughed up at intervals. She had pricking, tingling sensations in the hands and arms, and increasing difficulty in articulation. She grew considerably weaker, and had choking spells; the pupils were about equal. The enema was not retained this afternoon.

March 11th. Nutrient enemata were retained with difficulty, some not at all. Amount of urine in twenty-four hours, thirty-five ounces.

March 12th. Died.

Autopsy March 14th, 1898.

Body of a woman about 28 years of age, well developed, and nourished. Rigor mortis present.

Spinal cavity alone opened.

Cord on examination showed nothing abnormal. Hardening in formalin.

Microscopical examination: Sections from the lumbar enlargement show a marked dilatation of the blood vessels of the meninges, but without hemorrhagic extravasation. The nerve roots, in sections stained by Marchi's method, show diffuse degeneration of fibers, which is fairly extensive, and involves perhaps one-third of the fibers. This degeneration is somewhat more marked in the anterior than in the posterior nerve roots. The blood vessels in the nerve roots show the same marked dilatation and congestion which is present in the meninges

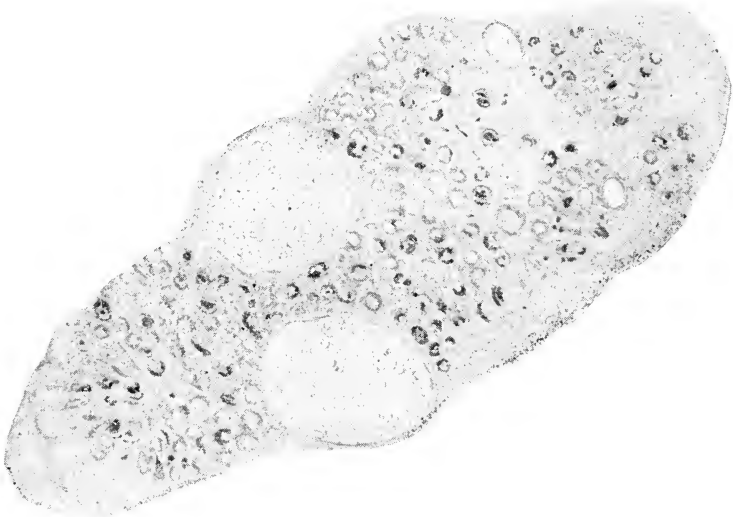
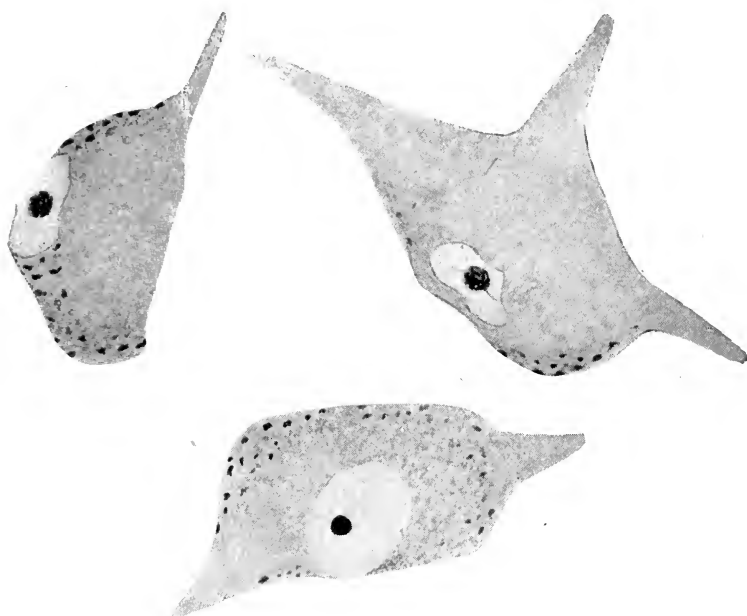


Fig. I. Low power. Zeiss, AA obj. No. 4 ocular. Nerve root, lumbar region, Marchi's method. Shows enormously dilated blood vessels, and degenerated nerve fibers.

with some hemorrhage among the nerve fibers. The blood vessels of the cord itself are also greatly dilated, particularly in the region of the anterior horns. A few of the vessels retain their normal size. There are no hemorrhages into the substance of the cord, nor is there any purulent infiltration. Sections stained by Marchi's method show a diffuse degeneration all through the white substance of the cord, and in the commissural fibers of the gray matter. Many of the nerve sheaths show the presence of fat, and sometimes they are en-

tirely replaced by fat. Occasionally the axis cylinder is replaced by fat, though this occurs less frequently than the degeneration of the myelin sheath. Rarely, both the axis cylinder and the sheath have undergone degeneration. The large nerve cells of the anterior horns are not decreased in number. They show no distinct fat globules, but in many of them there is a



Figs. II, III and IV. High power. Zeiss, oil immersion 1-16 obj. No. 4 ocular. Nerve cells from anterior horn of lumbar region. Nissl's stain, showing dislocation of nucleus, disappearance of nuclear membrane and destruction of protoplasmic granules of cell bodies. The nucleus in one of the figures appears to project beyond the cell wall.

single large group of pigment granules. The amount of pigment in these cells is greater than is normal at the age of the patient. In sections stained by methylene blue and eosin, and by Nissl's stain, few of the nerve cells appear normal. In most sections two or three cells with distinct granules are all that can be found, the others showing marked changes in the granules and nuclei. In many of the large cells which are cut through the processes, no nucleus or nucleolus can be found, though sometimes there is to be seen in the cell body a clearer spot, which evidently represents the location of the nucleus.

In other cells the nucleus stains faintly and homogeneously by Nissl's method, the nuclear membrane is broken or absent, and the nucleolus is faintly stained. In other cases the nuclear membrane is indistinct, the nucleus clear, however, while the nucleolus is larger than normal, stains a purplish color, and is vacuolated. In almost all the cells where the nucleus is seen it lies not in the center of the cell body, but is dislocated towards the periphery. Occasionally it lies so close to the cell wall that no protoplasm can be seen between it and the cell wall, and at times it even forms a projection in the cell wall, as though it were being extruded. In most of the cells spoken of above in which no nucleus can be seen, the clear area within the cell body, which represents the nucleus, lies excentrically. The protoplasmic granules of the cell body, even when present, do not stain sharply, but are ragged in outline, and vary a good deal in size. Occasionally a cell is found which has practically normal Nissl granules. In most of the cells, however, these granules cannot be made out at all, but the cell protoplasm stains homogeneously, or is filled with fine granular dust, with a few granules at the periphery of the cell. In the sections stained by Nissl's method, many of the cells show clear, yellowish areas in the cell body, caused by the presence of pigment granules. The dendritic processes of the cells are not broken, but can be traced as far as is usual. The granules in these processes, however, are fewer than normal; but occasionally they appear distinct, and of normal size.

The cells of the posterior horns of gray matter show a few granules at the periphery of the cell, and the nucleus and nucleolus are well preserved; the cells in general showing very little change.

In the dorsal region of the cord the same general changes are seen, particularly the engorgement of the blood vessels. By Marchi's method the same diffuse degeneration in the white matter is found. The cells of the anterior horns are as a rule more normal in appearance than those in the lumbar region. The nucleus and nucleolus show only rarely any marked dislocation. In some of the cells the nucleus has taken a faint blue tinge. The nucleoli are irregular in outline. The protoplasmic granules in many of the cells are normal, while in other cases they are fewer in number than normal, being seen chiefly at the periphery of the cell, while the cell body is stained faintly and homogeneously. The cells of the column of Clarke have fewer protoplasmic granules than normal, and these are chiefly at the periphery of the cell, but the changes are not marked.

In the cervical region there are found the same lesions in the white substance of the cord, and in its membranes, that were seen lower down. There are also degeneration of nerve cells, which are of the same character as those seen in the lumbar region, and fully as intense, but a smaller proportion of nerve cells are affected.

Sections from various levels, stained by Gram's method for organisms, failed to show their presence.

A cross section of the sciatic nerve, treated by Marchi's method, shows extensive fatty degeneration of the myelin sheaths of a good many of the fibers. The nerve fibers appear somewhat swollen, and where no fat is seen, they are exceedingly granular, and the axis cylinders cannot be made out. In

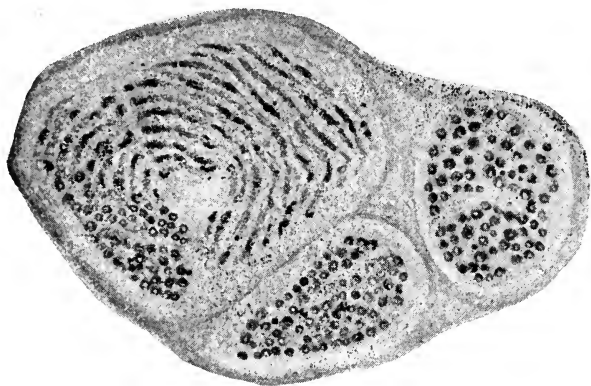


Fig. V. Low power. Zeiss, AA obj. No. 4 ocular. Portion of cross section of sciatic nerve. Marchi's method. Degenerated nerve fibers.

the longitudinal sections of the same nerve the osmic acid has penetrated poorly, but in the places where it has penetrated the myelin sheaths show an irregular, but extensive degeneration, which has affected most of the individual fibers.

#### Anatomical Diagnosis:

Parenchymatous degeneration of nerve cells of anterior horns, with destruction of protoplasmic granules, and nuclear changes.

Parenchymatous degeneration of anterior and posterior nerve roots, and of peripheral nerves.

Dilatation of blood vessels of anterior horns of the cord, of the meninges, and of the nerve roots.



To recapitulate, a fairly healthy young woman was suddenly attacked with weakness and later complete paralysis of the legs, extending to the arms, eyes and throat, with some involvement of the sphincters, slight disturbances of sensibility (pain, tenderness and paresthesia), loss of reflexes, and diminished reaction to faradism; the trouble resulting fatally in fifteen days. The autopsy showed degeneration of the nerve cells in the anterior horns, degeneration of the anterior and posterior nerve roots, and degeneration of the peripheral nerves, with dilatation of the blood vessels in and about the cord.

CASE II. For permission to report the second case, we are indebted to the kindness of Dr. Henry Jackson, in whose service the patient was at the City Hospital.

A. M., twenty-four, single, a laborer, a native of Cape Breton. His father died of rheumatism, his sister of phthisis. The patient had always been in good health and had rarely indulged in alcohol.

On the 18th of September, 1896, he was taken with severe headache, nausea and vomiting. The next morning he felt numb and weak and noticed a partial loss of power in both legs below the knees; he was able to go about the room with difficulty. The loss of power and numbness steadily increased and the next day he was unable to get out of bed, and felt feverish. There was no pain except when he was raised to a sitting posture, when there was some pain in the back. The appetite was poor; the bowels acted normally, but on the 21st, when he took a dose of salts, and on the 22d, when he was admitted to the hospital, he had an involuntary movement. There was no retention of urine. He was well developed and nourished, with an apathetic expression. The conjunctivæ were slightly injected; the pupils were equal and reacted normally; the pulse was regular and of good strength and volume; the tongue was protruded straight. Examination of the chest and abdomen was negative, except that the abdominal muscles were found to be weak; the abdominal reflexes were present; there was no tenderness of the back. The arms were normal. There was marked weakness of both legs; he was able to move the knees and feet only very slightly. The sensibility to pain was considerably diminished; the sensibility to touch and muscular sense was normal. The cremasteric reflexes and the knee-jerks were absent, the plantar reflex diminished; the muscles of the legs were flabby; the urine was negative. The temperature was 103° F.

On the 23d there was retention of urine with considerable pain so that he had to be catheterized. The weakness increased so that he could not turn in bed, and that day he had an involuntary movement of the bowels. The temperature was lower.

On the 24th he was more apathetic; the left side of the mouth drooped; he felt weaker; there was no pain, and sensibility to touch was normal.

On the 25th the temperature had fallen to normal, where it remained. The plantar reflex was absent; the arms were very weak; the left mouth drooped still more; he vomited and had many loose and involuntary stools. A lumbar puncture was made; one drop of clear fluid and a little blood were obtained, but no pathogenic organisms were found. He still had to be catheterized.

On the 29th he seemed brighter. An ophthalmoscopic examination was negative. He was able to pass urine voluntarily, but the stools were still loose and involuntary.

On the 3d of October there was so much pain in the legs as to prevent sleep. The appetite was good, but he was costive.

On the 9th, sensibility was still normal; there was no nerve tenderness. The paralyzed muscles were soft and flabby; the abdominal reflex was lost.

On the 13th he began to gain strength in the arms and gradually recovered control of the bowels, although very costive; he also had much rectal pain; there was complete loss of knee-jerk, complete loss of power and marked atrophy of the legs, and he could turn in bed only by the help of his arms. He required a daily enema. This condition remained until the 10th of November, when his friends took him to his home in the Provinces. Nothing has been heard from him since.

To recapitulate, a healthy man is suddenly seized with weakness of the legs, increasing to complete paralysis and extending to the abdominal muscles, the arms and the left side of the face, with loss of reflexes, loss of control of the sphincters, and hypoalgesia with no hypoesthesia. Later on there was marked muscular atrophy, and recovery was incomplete.

CASE III. The following case was seen in the service of Dr. C. F. Folsom, at the City Hospital, to whom we are indebted for permission to report the case.

William I., a colored laborer, single, aged twenty, a native of Massachusetts, was admitted to the hospital the 2d of November, 1898. The family history was negative. The previous

history was good, except for some muscular rheumatism in the right leg. He was a man of good habits. On the 17th of October he was in excellent health, except for some slight pain in the lumbar region, which he had had for two years. On the 19th, he woke with a heavy headache, general malaise and loss of appetite. The pain in the lumbar region was worse and he could not lie in any position that would relieve it. There was no pain or tenderness in the legs or excessive sweating. The legs began to feel numb, the sensation beginning at the tips of the toes and progressing upward daily. He began to lose power in both legs at the same time, the trouble rapidly increasing; the muscles of the trunk soon became affected; the respiratory muscles followed, so that he could not take a deep breath, and when he tried to do so the air seemed suddenly forced out; the arms also had grown steadily weaker. Just before admission to the hospital, he had some pain in the sternomastoid muscles. On the morning of admission, he found he could not swallow toast. There was no trouble in articulation, and no fever. He had normal control of the sphincters, but the bowels had not moved for three days. On admission, he was found to be well-developed and nourished; the mental faculties clear; the speech not affected; the tongue was protruded straight; the pupils were equal and reacted normally; the pulse was rapid, regular, of good volume and tension. Examination of the chest and abdomen revealed no abnormalities in regard to the internal organs; the knee-jerks were absent; the cremaster and plantar reflexes diminished; the sensibility to touch and temperature was everywhere normal; there was no hyperesthesia or tenderness over the nerves. He could flex the legs upon the thigh and the thigh upon the abdomen very feebly and with great effort. He could not raise the body while lying in bed. The hand-grasps were very weak. He could turn with much difficulty to one side—better to the right than to the left—and he could move the head without difficulty. The voluntary movements of the facial muscles were normal; no noticeable atrophy could be perceived. The extremities were cold. On the 6th, the weakness had increased, but he had had no pain. For the first time he noticed great difficulty in swallowing water, all that he took being regurgitated. The speech was thicker, slower and more inarticulate, and it was a great effort to speak. There was no special difficulty with the respiration. The masseters, even when he tried to close the jaws tightly, felt soft and flabby. There was double facial paralysis and anesthesia of the pharynx. The pupils were equal, but reacted sluggishly; he thought, however, that he could move the toes somewhat better. The weakness steadily

increased until, on the 10th, it had become almost impossible for him to swallow any food and it was decided to resort to tube feeding. There was not much difficulty, however, in respiration; the sensibility was unimpaired. The electrical reactions were unfortunately not tested. On the 10th he began to improve, and on the 11th, he was able to swallow a little food. The speech was less thick, but he was unable to close the eyelids or to pucker up the lips. There was no loss of control of the sphincters. He had some pain in the muscles after lying in one position, but he was able to move the toes slightly. The temperature during this period was, for the most part, normal, although occasionally, in the afternoon, there was an elevation of one or two degrees.

On the 15th the gain had become marked; he could move the legs in bed, especially in extension; he had no trouble in swallowing; the grasp of the hands was stronger; he could close the eyelids and also pucker up his lips to whistle; the voice was not much affected, and the masseters felt firmer. From that time he gained steadily.

On the 23d a blood-count showed 7,600 white corpuscles.

On the 27th of November he could whistle, close his eyes tightly, and he sat up in a chair by the bed. He had some pain in the left calf.

On the 3d of December he could sit up straight in bed and get from the chair into bed without help.

On the 9th of December, the calves were still flabby, but the arms were of good strength.

By the 16th he was able to walk fairly well, although the legs had not regained their full size, and on the 22d he was able to leave the hospital. He has since made a complete recovery.

To recapitulate, a healthy young man is suddenly attacked with weakness of the legs, increasing to complete paralysis and extending to the abdominal and respiratory muscles, the muscles of the face and the muscles of mastication and deglutition. The reflexes were lost. The sensibility was not markedly impaired. There was later some muscular atrophy with complete recovery.

These three cases present three different terminations of Landry's paralysis and also raise certain questions as to the clinical features of the disease. The pathological lesions show the close connection of the affection with acute multiple neuritis and with acute poliomyelitis. Pathologically, it is both;

clinically, however, it differs somewhat in its manifestations from the ordinary type of either disease. The course of ordinary anterior poliomyelitis is quite different; beginning as Landry's paralysis may, with signs of acute febrile disturbance, it affects a considerable number of muscles at once, and is not progressive in character; the abdominal and respiratory muscles and those supplied by the cranial nerves are seldom affected and the sphincters remain intact. Sensory disturbances, except some painful sensations, are lacking. In neuritis, on the other hand, sensory disturbances are more marked. The trouble is less often progressive, and the cranial nerves are usually spared. In neither affection do we note the steady progress as in Landry's paralysis, from the feet upward until the cranial nerves are involved. Whether Landry's paralysis may extend downwards, as it is said to have done with Cuvier, requires further evidence.

Although the poison which produces Landry's paralysis is, like lead, selective, affecting the motor neurone, we must remember that sensory disturbances, amounting to anesthesia of the feet and finger-tips, were present in Landry's original case. The sensory disturbances are much less marked, but they are by no means uncommon, and the presence of degeneration in the posterior nerve roots in the case presented to-day gives a partial explanation. The neuritis which is so often present may affect sensory fibers as well as motor, although the affection of the motor fibers predominates.

The disturbance of the sphincters, noted in two of the cases, is not usual, but it may be explained in part by the weakness of the abdominal muscles preventing effective straining. It has been met with, although rarely to a marked degree, in cases otherwise presenting the symptoms of Landry's paralysis so that its presence can hardly be regarded as disproving the diagnosis.

In the two cases that recovered, there was considerable muscular atrophy, and in the fatal case there was a diminished reaction of the muscles to faradism. One of the striking features of Landry's paralysis, as given by Landry himself, and as restated or confirmed by the majority of writers since, is the existence of complete paralysis without muscular atrophy and

without changes in the electrical reactions. Westphal<sup>18</sup> in 1876, defining Landry's paralysis, stated that it was characterized by its progressive ascending course with fatal termination, by the fact that the paralyzed muscles retained their electrical excitability intact, and by the negative results of autopsy. The progressive ascending course is essential, but, as these two cases show, the termination is not inevitably fatal and the results of autopsy are not negative. The question of atrophy and electrical changes is still open.

With the establishment of the anatomical changes in Landry's paralysis as a degeneration of the peripheral motor neurone, we must admit *a priori*, if our modern notions as to the symptomatology of nervous affections be correct, that muscular atrophy and altered electrical reactions will inevitably result. Muscular atrophy and marked electrical changes, however, require a certain time for development, and in Landry's paralysis the patient may be dead before they ever become marked. The ordinary methods of clinical examination, moreover, do not favor the detection of slight changes in electrical reactions, especially quantitative estimates. In a considerable number of cases where the paralysis was acute, ascending and progressive, especially in cases that did not end fatally, muscular atrophy and electrical changes have been noted. In Burghart's case already cited,<sup>19</sup> which recovered from Landry's paralysis to die of tuberculosis, reaction of degeneration was observed. It is easier to believe that slight electrical changes have been overlooked than that a pronounced degeneration of the peripheral motor neurones can exist without some changes. In severe cases that recover, therefore, atrophy and electrical changes are to be looked for, but many cases die before atrophy becomes marked.

#### DISCUSSION.

Dr. F. W. Langdon said that, with reference to the sensory symptoms in Landry's paralysis, he had had the opportunity of seeing a case within the past year in which the diagnosis was confirmed by the fatal outcome. It was in the person of a man weighing over 200 pounds, who was connected intimately with

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<sup>18</sup> Westphal, Archiv für Psychiatrie, vi., 765, 1876.

<sup>19</sup> Burghart, *art. cit.*

the brewing business and drank freely of beer. He had for three or four days before Dr. Langdon saw him numbness in his fingers and hands. He had no symptoms in the legs, and rode about, standing on the platforms of cars because he smoked. When Dr. Langdon saw him he had this distinct numbness in the arms, but could feel the ordinary touch of the hand. There was a very suspicious weakness of respiration, and the development of Landry's paralysis was feared. There was also tenderness on pressure over the principal nerve trunks, but no spontaneous pain. A guarded prognosis was given and the family was frankly informed that the outcome could not be foretold, and that if the disease ascended any higher it would probably be fatal. The man died two days later. The probabilities are that the symptoms of numbness, etc., are so slight as compared with other symptoms that they are often overlooked.

Dr. C. E. Riggs stated that he had under his care a case of multiple neuritis that was rather anomalous in its development. For the first three or four days the patient complained of weakness in the lower extremities, extending later to the upper extremities, and he was brought into Dr. Riggs' office absolutely unable to make any voluntary motion. Perhaps a week afterward he commenced to suffer severe pain, and the evolution of the case made it clear that it was a form of multiple neuritis. Another point that quite impressed Dr. Riggs was that at one of his visits he observed that the patient had a very rapid pulse, with perfect regularity of the respiration and heart beat. In the evening these symptoms increased and a condition of nervous shock developed, so that Dr. Riggs thought that the man would not live through the night. He was placed under opiates and began to rally, and is now getting along very nicely.

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- II. ALBUMINURIE POST-PAROXYSMIQUE DANS L'EPILEPSIE CONVULSIVE (Post-Paroxysmal Albuminuria in Convulsive Epilepsy). M. Lannois and L. Mayet (*Lyon Médical*, 31, 1899, p. 365).

In a study of fifty patients involving about four hundred urine analyses, the authors found albumin in the urine after fifty-five per cent. of epileptic paroxysms and suggest that its presence may be of signal value in excluding simulated epilepsy and hysterical convulsions. After mention of several explanations of the presence of albumin, they conclude that it is due to circulatory disturbance induced by the fit and note with Voisin and Péron that it is the patients who become cyanotic in the attack who afterward void albuminous urine. This post-epileptic albuminuria is transitory and in direct relation to the intensity of the asphyctic stage of the paroxysm.

PATRICK.

## A CASE OF HEMATOMYELIA.\*

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Cases of hematomyelia in which the lesion is exceedingly small and circumscribed, and in which the clinical phenomena are carefully studied and noted, are sufficiently rare and important to be worthy of a special detailed report. The following case was one of this type, as the patient was under observation during almost the whole short period between the accident and her death, and the lesion has been studied and illustrated with care. The case was one in which the only lesion was situated in the substance of the cord, for, although the accident had been one of great violence, no injury had been sustained by the vertebræ or the membranes. It is thus representative of the pure type of hematomyelia—a rare type, whether due to trauma or to primary disease of the blood vessels. It has especial importance as throwing some light on the subject of spinal localization, and also as illustrating a class of cases in which the temptation is always great to invoke the aid of surgery—an aid which must necessarily always be in vain.

J. C., a colored woman, aged 53 years, in good health, without any personal history of alcoholism or syphilis, fell in the dark down a flight of stairs. She could not tell how she struck, or where she was injured, but she was immediately paralyzed in her arms, body, and legs. There was no loss of consciousness. The accident occurred on April 9, 1899, and the patient was taken to a large general hospital, from which she was discharged unimproved in a few days for lack of accommodation. There was no history of her condition during these few days, except that she was paralyzed as above mentioned. On the 14th (five days after the accident) she was admitted into my wards at the Philadelphia Hospital.

On admission her condition was as follows:

*Motion:* There was complete paralysis in all the extremities. Power was retained in the trapezius, sternocleidomastoid, and sternothyroid muscles, and in the diaphragm. The muscles of the chest were paralyzed.

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\*Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

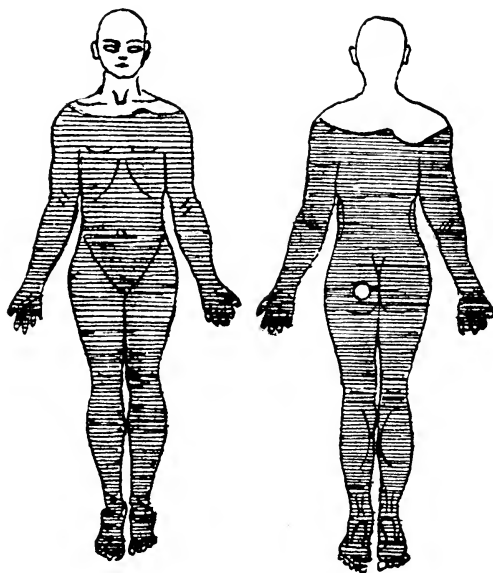


*Sensation:* Below a line indicated in the diagrams (Figs. I and II) all modes of sensation were absolutely lost.

*Reflexes:* There was a slight biceps and triceps jerk on the left side. Both knee-jerks were exaggerated. No ankle-clonus. No plantar reflex. This preservation of the knee-jerks is noteworthy.

*Respiration* was diaphragmatic and irregular. Inspirations were gasping, and not complete. There were no movements of the chest walls.

The *bladder and rectum* were paralyzed, and there was consequent incontinence of urine and feces.



Figs. 1 and II. Hematomyelia. Total anesthesia below the neck. Bedsore on right buttock.

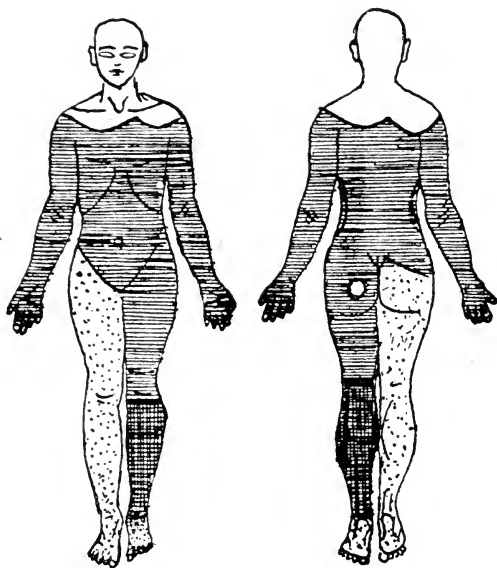
*Cerebrum:* The mind was clear, the patient responding promptly to questions. The tongue was protruded in a straight line.

*Eyes:* There was an old, probably congenital, double external strabismus. It was in no wise related to the patient's condition. It has been made the subject of special study by Dr. Oliver.

There was no spinal tenderness or deformity. There was no pain in the neck, and the movements in the neck, both active and passive, were good.

In general, the patient's physical health was good. She was a large, robust woman, without any disease of the heart, lungs, kidneys, or abdominal organs.

April 15. The patient said that she felt better, but her condition was practically the same as on admission. Careful tests were again made. The area of anesthesia had not increased. Retention of urine and feces was present during the night. The ciliary reflex from irritation of the skin of the neck was absent. An examination of the eyes was made by Dr. Oliver, who studied the congenital strabismus already

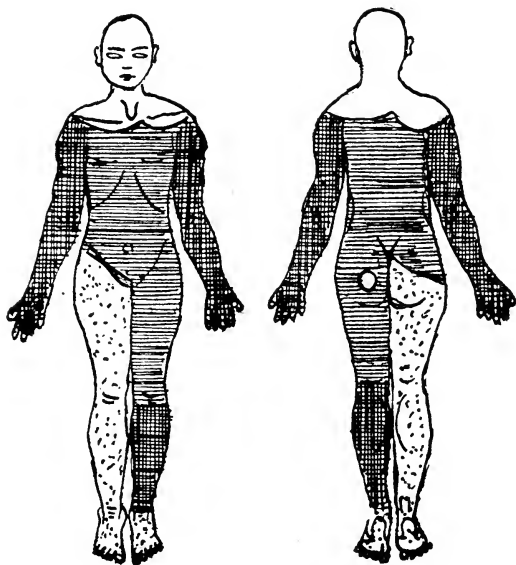


Figs. III and IV. Hematomyelia. Dotted area shows return of sense of pain. Crossed area shows return of sense of heat and cold.

noted. No affection of the eyes was observed by him that could be ascribed to the accident, except that there was no sympathetic mydriasis in either eye; but there was spastic myosis, although the pupils responded promptly to light and on accommodation.

April 16. About 4 P. M. the patient complained of pain in her knees. She breathed better. The bowels were moved by enema. Retention of urine continued. A bedsore on the left buttock, which had begun to form before the patient's admission, had increased in size since the preceding day.

*Motion:* Paralysis in all the extremities was still absolute. Pricking the leg caused it to be drawn up, but the patient had no control over it. *Sensation:* The sense of position had returned in the lower limbs. Pain sensation on pricking with a pin had returned in the right lower limb and left foot; also pain from pinching the muscles was noted in the same areas and in the left leg. The sense for heat and cold was noted about the right knee, but tactile anesthesia was still complete everywhere below the original boundary line. (See Figs. III



Figs. V and VI. Hematomyelia. Dotted area shows return of sense of pain. In crossed area sense of pain on pinching skin and muscles; also sense of heat and cold in limited spots.

and IV.) Later the pain sense returned in the upper limbs. (See Figs. V and VI.)

April 17. Breathing still better. Bedsore increasing superficially. Sense of position had returned partially in the upper limbs, and pain was caused by pinching the muscles in the arms. The only return of tactile sensibility was in the left foot, as noted yesterday, except that at times the patient could locate a pin prick in the left arm. No return of motion. The improvement in the breathing and the slight return of some of

the modes of sensation caused the case to have a slightly better aspect on this day.

April 18. There was practically no change in the patient's condition on this day. In the evening Dr. La Place and Dr. Frazier, surgeons to the hospital, examined the spine, but they could not detect the presence of any fracture; hence, surgical interference was decided against.

April 19. The patient's condition was worse. The kneejerks were noted to be *absent* for the first time. Incontinence of urine continued. The breathing had again become difficult. There was a slight bleeding from the right nostril, which, however, did not continue. The patient was somewhat stuporous, probably because of a rising temperature and deficient aeration of the blood. *All* sensation below the original line was again abolished, as on the patient's admission. There was now noticed for the first time *moist skin above*, and *dry skin below* this line. During the day the patient progressively failed. The temperature rose rapidly until it almost reached 106°. The lungs filled with moist râles. The breathing became more and more difficult from gradual paralysis of the diaphragm. Vomiting occurred at 6 P. M., and the patient died at 7 o'clock. She had survived the accident ten days. The notes of the case were taken by Dr. G. E. Pfahler, the resident physician.

*Autopsy:* Remarkably little evidence presented itself at the autopsy of severe injury to the structures of the neck. There were no wounds or ecchymoses in the soft structures. There was no deformity or swelling of the neck. The cervical vertebræ were uninjured; there was not the slightest dislocation or fracture, as determined by a searching examination. Within the spinal canal everything seemed normal at the first cursory view. The membranes presented nothing abnormal; they were not the seat of changes of any kind, such as would be caused by tearing or by inflammation. There were no meningeal hemorrhages. The cord itself at first, as viewed *in situ*, did not appear in any way changed. On closer inspection, however, even before it was taken out, it was seen to present a somewhat swollen appearance in about the mid-cervical region, and to be of an abnormal pallor at this point. The cord was removed without difficulty and without injury, as it was not perceptibly softened or adherent at the point of swelling. After its removal the underlying membranes and bones were carefully examined and found to be in every respect normal. On laying the cord open by making transverse sections through the affected portion, it was readily seen that it was the seat at this point of a small hematomyelia. To

the naked eye the center of the cord seemed softened, and to contain quite a perceptible quantity of liquid blood or bloody fluid. This extravasation of blood appeared on gross inspection to occupy rather the central gray matter than the surrounding white substance, and to be rather larger than it proved to be eventually under the microscope. It was also seen later under the microscope that it was not so much the gray matter as the white that was involved. Its greater apparent extent in the fresh tissue was doubtless due to some degree of edema and engorgement. The general impression

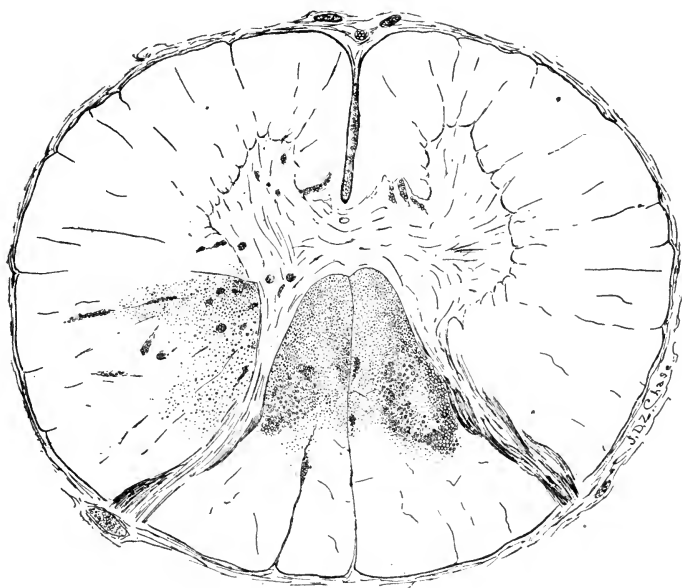


Fig. VII. Hematomyelia. Fourth cervical segment.

made upon all who saw the specimen in its fresh state was one of surprise that so little injury had been done, and that such a small lesion was capable of causing such speedy death. None of the other organs or tissues of the body presented any evidence of injury, or of any disease that is here noteworthy.

The microscopic sections of the cord have been kindly made by Dr. Wm. G. Spiller, and present the following appearances:

The lesion is located entirely above the *sixth* cervical

segment. The sixth segment presents nothing abnormal, except some rather doubtful appearances of degenerative changes about the periphery of the cord, and which are seen all through the higher segments which were the seat of the hemorrhage. These changes may have been caused by edema of the cord. There is no descending degeneration in the lateral columns at this point.

At the *fifth* cervical segment is seen the lowermost limits of the hemorrhage. These consist of three small areas located entirely in the posterior columns at their anterior limits. The larger of these is in one posterior column in direct contact with the gray matter; the other two, smaller in extent, are in the other columns, also in its anterior part. The surrounding white matter is here apparently normal, as the small hemorrhagic foci are sharply outlined. The gray matter is uninjured.

At the next level just above, and consequently involving the *fourth* cervical segment, the hemorrhage is seen in its greatest extent. It involves almost the whole of the anterior half of both posterior columns. It is symmetrically arranged, as represented in the diagram (Fig. VII.). Extravasated blood can be seen occupying a point in each column, and the white matter is extensively broken up. Dilated blood vessels are also present in and around the injured tissue. The gray matter is apparently quite exempt. The white matter, however, shows evidence at this level of injury in other regions, though not of hemorrhage. About the periphery and in the left lateral tract there is degenerated and swollen tissue, evidently the result of bruising and engorgement of the spinal medulla. The lateral tract is especially injured, and may even contain some extravasated blood near the posterior horns. This posterior horn is thus involved possibly to some extent in the lesion, though its tissue does not seem to be deeply injured.

In the *third* cervical segment the lesion occupies almost the same limits as in the fourth, although it is evidently not quite so destructive. Other areas of degeneration are here more intense, especially the left lateral column, with the contiguous ascending cerebellar tract; also at the periphery of the anterior columns. In many scattered regions of the white matter in fact, at this level, although there is not deep degeneration, there is evidently such a condition as might result from bruising and swelling of the tissue; hence, it is evident that the cord was injured more extensively than is merely represented by the hemorrhage in the posterior columns.

At the *second* cervical segment the lesion is still quite extensive in the posterior columns, but this is apparently its uppermost limit, and it is not so extensive as below. The other

changes in the white matter are about as at the lower levels, although not so intense. The lesion was not traced above this level. Its whole vertical extent may thus be said to be from the fifth upward to about the lower part of the second cervical segment. The lesion was thus in both its vertical and horizontal extent a comparatively limited lesion, consisting of a small hemorrhage in the anterior portions of the posterior columns, and of bruised and swollen tissue in these columns and also in some other comparatively limited areas of the white matter. With the thionin stain the multipolar cells in the anterior horns in this region show distinct chromatolysis. Many of them are much swollen and misshapen, and the chromophilic substance is broken up and in some instances entirely destroyed.

*Analysis of the Symptoms:* The clinical phenomena in this case are in the main clearly and logically related to the microscopic findings.

The *motor* symptoms indicate very clearly a lesion as high at least as the third cervical segment, though not completely destructive of this segment. Thus the trapezius and the sternomastoid muscles, the motor nerve of which probably has some of its cells of origin in the third segment, were not paralyzed, but all the muscles of the body below them, except the diaphragm, were completely paralyzed. The exact spinal center for the phrenic nerve is a question of some uncertainty. Anatomists even differ as to the superficial origin of the nerve; thus Gray says that it arises from the third and fourth cervical nerves, while van Gehuchten says it arises from the fourth and from an anastomosing branch from the fifth. Most clinicians in their diagrams locate the spinal origin in the fourth and fifth segments, but this localization is probably in most cases not indicative of very exact knowledge. The more exact tables founded upon the observations of Mills, Starr, Thorburn and others place the center for the phrenic nerve in the second, third and upper part of the fourth cervical segments. My case is confirmatory of this localization as a whole. The phrenic nerve was evidently involved, as shown by its embarrassed action, and yet its function was not totally abolished, as shown by the fact that the patient's life hung on it for ten days. Her death, however, was eventually brought about by progressive failure of this nerve—a termination which I have seen in

other cases of cervical injury, and which is due possibly to the gradual involvement of the motor cells and fibers of this nerve in a progressive degenerative action. In the case here reported, the third and fourth segments were extensively injured, but not totally so.

The *sensory* symptoms were indicative of a lesion as high at least as the third cervical segment. The fourth segment was undoubtedly invalidated, as it supplies sensation to the outer side of the arm, which in this case was anesthetic. The partial return of sensation for pain, heat and cold was an interesting phenomenon, and is probably to be explained by the fact that the central gray matter was not so much injured as the white. This is in accord with the view that the tracts for these modes of sensation pass through the gray matter, and then pass upwards through the anterolateral columns, which in this case were the least involved. The only return of tactile sense was in one foot, which may be explained by the fact that the tactile fibers for the lower limbs pass in the neck by the posterior parts of Goll's columns, which in this case were not injured.

The state of the *reflexes*, especially the knee-jerks, was a point of interest. These were not abolished, but exaggerated. It is well known, since Bastian originally called attention to the fact, that in total transverse lesions, especially when accompanied with shock, as in traumatic cases, the knee-jerks are at first abolished. All the conditions in my case seemed to indicate that the knee-jerks should be abolished. The fact that they were preserved and even exaggerated, led me to the belief at the bedside that the patient did not have a total transverse lesion, although the motor and sensory paralysis was at first strongly indicative of such a lesion. This preservation of the reflexes may be associated in some way with the preservation of the gray matter.

The hyperidrosis *above* and anidrosis *below* the boundary line are worthy of special note. Irritation of the sympathetic system has been known to cause excessive sweating, and its paralysis the opposite condition. Unilateral hyperidrosis has been seen in cases of hemiplegia. I have reported a case of



unilateral facial hyperidrosis in a case of hemiplegia.<sup>1</sup> In the present case the inference is that the sympathetic fibers were irritated above and paralyzed below the seat of lesion.

#### DISCUSSION.

Dr. Spiller said that Dr. Lloyd had sent him a portion of the spinal cord for examination. The cord had been cut through in several places, and it was therefore difficult to determine the segments. The eighth cervical segment has a very characteristic form, and is easily recognized, although this fact does not seem to be generally known. The anterior horns of this segment are very concave in their anterior portion. The necrotic areas in Dr. Lloyd's case were central, and involved the white matter more than the gray, and the nerve cells of the anterior horns in the injured portion of the cord showed distinct chromatolysis. Above the sixth cervical roots the segments could be determined only approximately.

Dr. Fisher said that Dr. Lloyd's paper had brought to his mind very clearly a patient who was under his observation last winter and recovered. A boy, while playing football, had his head driven forcibly down on his chest; he became unconscious and afterwards had paralysis from the neck down. One month afterwards he had complete inability to move the arms or legs and the sensory disturbance consisted in lowering, but not loss of all forms of sensations. The right arm and leg were more affected than the left, and showed considerable wasting. Gradually the condition changed, and he became more paralyzed on the left than the right side. The reflexes were very much exaggerated. He finally made a recovery with slight paresis involving the left lower extremity. Dr. Fisher came to the conclusion that the patient had some capillary hemorrhages in the spinal cord, probably in the lateral tracts. The case lasted a long time, and the symptoms were of as severe a character as would attend a graver lesion.

Dr. Patrick thought that these injuries to the cervical portion of the cord without fracture could not be very rare, because he had seen several of them, and in at least two cases had advised against operation, believing that the lesion was in the substance of the cord, perhaps principally in the gray matter, and the subsequent course of these cases seemed to justify that conclusion.

As regards the preservation of the knee-jerks in the case

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<sup>1</sup> Twentieth Century Practice of Medicine, Vol. IX. In this article a number of literary references are given.

reported by the President, Dr. Patrick thought that the preservation of sensation demonstrated that the section of the cord was not complete.

Dr. Onuf said he had had one case that was extremely instructive to him in regard to the sensory disturbances. It was a case of fracture of the vertebral column in the upper lumbar region, and there was absolute paraplegia. The lower limbs were completely paralyzed, but the sensation was preserved throughout in one extremity, although greatly diminished, and in the other extremity it was preserved in the thigh, but completely absent in the foot and leg. In that case the preservation of sensation led him to make a good prognosis, and the patient made an almost complete recovery.

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12. UEBER DIE BEDEUTUNG DER ZEHEN-REFLEXE (Concerning the Meaning of the Toe-Reflex). Martin Cohn (Neurologisches Centralblatt, July 1, 1899, No. 13).

This investigation was undertaken to find out the importance of Babinsky's "Phénomène des orteils," a reflex of the toes, caused by irritation of the sole of the foot. In normal cases a stimulation here is followed by flexion of the toe at the metatarsophalangeal joint. In injuries of the pyramidal tract, or when the same is made functionless by cerebral injury, an extension of the toe follows stimulation of the sole of the foot. Further, this phenomenon of extension is supposed to be one of the earliest symptoms of a lesion of the pyramidal tract, and it can be diagnosed from this symptom before any other signs appear. The following conclusions are noted by the author, as his results from the examination of normal and abnormal individuals: The extension movement of the toe is seldom found in persons with normal nervous systems. In infants as a result of the lack of myelin sheaths in the pyramidal tracts, according to Babinsky, the toe extension should always follow. In most cases, the author found this to be true; in other cases, however, no toe reflex was found at all. In abnormal cases the observations were made mostly on lesions in the pyramidal tracts. In two cases of amyotrophic lateral sclerosis, a clear extension of all toes was found. In two cases of spastic spinal paralysis the same result was observed. In another case of this disease a flexion of the second and fifth toes and a weak extension of the first were noted, and in another case no reflex at all was found. In cases of fresh apoplectic paralysis, where according to Babinsky, this sign should be seen first, the author found no reflex at all. In two cases, an extension of the first toe was present while examination made a few days later showed no reflex at all. In two cases of cerebral tumor which had extended to the internal capsule, a definite extension movement was found on the side opposite to the tumor. In a case of total hysterical paralysis of one of the lower extremities, a toe extension movement was found on the paralyzed side, which was contrary to Babinsky's findings.

The author comes to the following conclusions: In the majority of all persons an irritation of the sole of the foot is followed by flexion of the toes. In lesions of the lateral tract of the spinal cord of an organic character, an extension reflex is to be observed. In no way, however, can this phenomenon be regarded as a certain pathognomonic symptom for the recognition of such diseases. SCHWAB.

## A CASE OF TUMOR AT THE BASE OF THE BRAIN IN THE PONTINE REGION.<sup>1</sup>

BY JAMES HENDRIE LLOYD, A.M., M.D.,  
NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

The patient from whom this specimen was obtained had the following history: A. T., female, aged 48, had good personal and family antecedents. About January, 1898, she began to have nausea, vomiting, and vertigo, without known cause. One day the vertigo became so intense that the patient fell from her chair, after which she noted diplopia with strabismus. Then began failing vision in the left eye. There was a history also of headache in varying intensity, and of inco-ordination of gait, so that she required to be assisted in walking.

On admission to the hospital, in March, 1898, the patient presented the following symptoms: Her gait was ataxic in character, and she had a tendency to sway and pitch to one side; this was not, however, a typical forced movement. The left eye presented slight paresis of the external rectus muscle, and the patient could not close this eye as firmly as its fellow (combined involvement of the sixth and seventh nerves). There was no hemiplegia or other paralysis, nor any involvement of sensation at this time. The hearing in the left ear was impaired. Dr. Oliver found paresis of *both* external recti, more marked on the left side; also, marked neuro-retinitis, with numerous hemorrhages in both neural and retinal tissues.

The further progress of the case was marked by increasing paralysis of both sixth nerves and of the left seventh nerve. Hearing in the left ear gradually failed, until there was complete loss of power in the left eighth nerve. The optic neuritis progressed until it caused total blindness in both eyes. The third and fifth nerves never became involved. There was never any loss of power in the arms or legs, showing that the motor tracts in the pons and medulla were not involved. Neither was there any loss of sensation in any region of the body. The progress of the case was slow and long-continued. The affection of the gait became so great that the patient was soon confined to bed. She lived for more than one year, dying eventually of exhaustion. Towards the end she was in a stuporous condition most of the time, from which she could be only partially aroused.

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<sup>1</sup> Paper read and specimen presented at the annual meeting of the American Neurological Association, Atlantic City, June 14 and 15, 1899.

A diagnosis of tumor of the pontine region was made. The involvement of both sixth nerves and the left seventh and eighth nerves, without implication of the motor or sensory tracts within the pons, indicated that the growth was on the outside of the pons, where it was found after death.

AUTOPSY.

*Brain:* Scalp and calvarium presented nothing abnormal. Dura was not especially adherent. No excess of fluid in subarachnoidal space. Small vessels of pia were injected; large vessels distended. Pia was transparent everywhere. Brain was flattened in vertical axis. Springing from the sulcus between the pons and cerebellum was a cone-shaped tumor, the apex of which projected forward to a point slightly anterior to the middle line of pons and reached to within 15 mm. of the origin of left crus cerebri. The apex of the tumor was rounded and shaped like a nipple. The base of the tumor was a little anterior to the middle of the left cerebellar hemisphere.

The tumor, which was about the size of a small hen's egg, was 3 cm. wide at its base. Its total length was 5.5 cm. Its greatest thickness was 3.3 cm. Laterally, toward the median line, the tumor pressed upon the medulla and the posterior part of the pons on the left side. The medulla was twisted in its longitudinal axis, and was directed at an angle of 45 degrees away from the median line, so that its left lateral aspect was turned obliquely upward, and in addition, was flattened from side to side. The tumor was sharply circumscribed, and could be shelled out from the cerebellum. It was covered by the pia in its posterior aspect, and possibly also anteriorly. It had an irregular, convoluted surface, and resembled brain tissue in appearance, and was firm in consistence. On section, the growth had a yellowish-red appearance, but here and there was marked by distended vessels and small hemorrhages. The seventh and eighth nerves on the right side were distinct. On the left side, in the region of the tumor, they were indistinct. There was marked softening of the pons, where the tumor infringed upon it, and for about 2 cm. forward.

*Base of Skull:* Tumor was adherent. Posterior part of petrous portions of temporal bone was eroded. The eroded cavity was 2 cm. (long) x 1 cm. (high) x 1 cm. deep. Central part of excavation corresponds to the position of the internal meatus. Bone was rough. Sharp ridges passed across bottom. Merely a shell of bone was left anteriorly. Cavity was filled with soft, red tumor tissue.

## ANNIVERSARY ADDRESS.

BY WHARTON SINKLER, M.D.

The address commemorative of the twenty-fifth anniversary of the association was delivered by Dr. Sinkler. He called attention to the fact that the American Neurological Association was the third of the national associations which had been organized. At the time of its formation there were but two neurological associations in Europe, both being in Germany. The first meeting was held in New York, June 2, 1875. Dr. Jewell, of Chicago, was the first president. There were thirty-five original members, and the limit of membership was fixed at fifty. A few years ago the limit was increased to one hundred, and at the present time there were ninety-seven members. After speaking of the advances made in neurological science during this period, and referring especially to the advance made in cerebral and spinal localization by American neurologists, he concluded with a list of books and monographs contributed by members of the association.

## THE NERVOUS EQUIVALENT OF FEVER.\*

BY HENRY S. UPSON, M.D.

Dr. Upson said that it was usually easy to distinguish organic from functional disease in the nervous system, but the problem became difficult when it was necessary to differentiate functional nervous troubles pure and simple from disease of other organs with nervous symptoms. After citing several illustrative cases, the following conclusions seemed to the speaker to be warranted: (1) Fever was a nervous reaction, usually to toxins; (2) fever was often accompanied by other nervous reactions; (3) other nervous reactions to toxins, whose form was largely determined by the predisposition of the patient, might occur without fever, and might replace it. These reactions might simulate very closely hysteria, mania, and melancholia.

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\*Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

## Society Reports.

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NEW YORK NEUROLOGICAL SOCIETY,

December 5, 1899.

The President, Dr. Frederick Peterson, in the chair.

### MULTIPLE SCLEROSIS WITH SYMPTOMS OF PROGRESSIVE MUSCULAR ATROPHY.

Dr. Graeme M. Hammond presented a case of multiple sclerosis occurring in a man, thirty-eight years of age, exhibiting symptoms resembling those of progressive muscular atrophy. This combination, he said, was quite rare. The patient was a locomotive fireman by occupation, and had a good personal and family history. About one month after an attack of the grip ten years ago his present trouble began. The first symptom had been a paralysis of the internal rectus of the left eye, and this had been followed by weakness of the left superior and the right internal rectus. At the present time, in addition to these symptoms, he had left hemianopsia. About one year ago he had had a sudden loss of smell on both sides. About six years ago he had begun to show symptoms of locomotor ataxia. The knee-jerks were absent. Last January he had noticed weakness in the little and ring finger of the right hand. This weakness had extended to the other fingers, and had been accompanied by atrophy. The atrophy was now well marked in both upper extremities, and fibrillar twitchings could be noticed in the affected muscles. These muscles responded slightly to the galvanic current, but not at all to the faradic. This history was very characteristic of multiple sclerosis. In 1897 a similar case had been reported in one of the German journals. In the case presented true intention tremor and scanning speech were not present. It had occurred to Dr. Hammond that the patient might be suffering from a progressive muscular atrophy engrafted upon a multiple sclerosis, but it was also possible that the sclerotic changes had taken place in an unusual position in the anterior horns. The optic nerves had remained normal.

Dr. B. Sachs thought that the case might be almost anything else than multiple sclerosis. This diagnosis did not seem to him justified in the absence of nearly all of the cardinal symptoms of the disease.

Dr. Joseph Collins regarded the case as one of locomotor ataxia associated with progressive muscular atrophy. It was possibly an example of syringomyelia and tabes, like a case he had had under observation for a long time.

Dr. George W. Jacoby agreed with the last two speakers, because

the symptoms in the upper extremities and the optical symptoms resembled those of a nuclear affection, while the symptoms presented by the lower extremities were like those of tabes. It was, of course, rather presumptive to make a diagnosis after Dr. Hammond had studied the case so carefully.

Dr. Frederick Peterson thought that this patient presented all the symptoms of locomotor ataxia. Atrophies of this kind were not very uncommon in locomotor ataxia. Several years ago he had exhibited to this society a case of typical locomotor ataxia with three symmetrical quadrants of vision lost, so that the person saw out of only one-quarter of each eye. He, therefore, looked upon the case as a locomotor ataxia presenting the unusual symptoms already cited.

Dr. Hammond replied that at first he also had looked upon this case as one of locomotor ataxia with ocular symptoms, but, on studying it more carefully, he could not find the slightest indication of syphilitic infection, the man having indeed been singularly free from previous illness of any kind. He was temperate in his habits, was not neurotic and presented an unusually good personal history. Again, his tabetic symptoms had not been at all prominent; the Romberg symptom was hardly noticeable, and there were none of the bladder or sexual symptoms of tabes. The fact that there had been lesions of the optic, the third and the olfactory nerve, coming on respectively at intervals of several years, he looked upon as proof that sclerosis had affected these different nerves. An involvement of the posterior columns in disseminated sclerosis was not at all uncommon. The atrophy in the hands was, however, decidedly unique.

#### INTRACRANIAL GROWTH.

Dr. Philip Meierowitz presented a man of thirty-eight years, who had come to him on December 1, 1899, complaining of amblyopia of the left eye. He was entirely blind in the right eye from an injury inflicted with a piece of steel. The disturbance of vision had first appeared in the summer of 1898, and had reappeared four months ago. The "blind spells" had come on several times a day, and had lasted about eight minutes each time. They had continued altogether for about six weeks, and had been unaccompanied by pain. A tremor in the right upper extremity had also developed about the same time as the trouble with the eyes. Jerking of the right arm had been quite marked at night. Vertical headache had been present, and quite troublesome some months ago, but had disappeared. About the middle of last July he had been seized with attacks of vomiting after taking food, and had improved under milk diet and the administration of iodide of potassium. About this time he had a sudden loss of consciousness, and again, two or three months ago, he had a similar attack. There was no distinct history of syphilis. Examination showed dilatation of the left pupil and good reaction to light, with absence of nystagmus. Tremor of the right arm was quite marked, and was aggravated by movement. His gait was good; the knee-jerks were exaggerated; there was no ankle-clonus. He had no sensory disturbances. Dr. Valk had examined the eyes with the

ophthalmoscope, and had found a papillitis. There was no mental dullness, and no impairment of the memory. The speaker expressed the opinion that the man had an intracranial growth, and he believed that its location in the cerebellum would account for most of the symptoms. He was inclined to believe that it was a syphiloma because of the effect of treatment with the iodide and the lack of definiteness regarding the absence of syphilitic infection.

Dr. B. Onuf said that the patient had been under his care at the time that he had received the iodide. He had then a staggering gait, a marked intention tremor, increased knee-jerks and ankle-clonus on the right side. His eye showed marked choked disk and a number of retinal hemorrhages. He had at first hesitated between a diagnosis of tumor and multiple sclerosis. Dr. Coffin had expressed the opinion that the shape of the hemorrhages—small and wedge-shaped—pointed rather towards syphilis. The man had then been put upon rapidly increasing doses of the iodide, and had improved promptly. The dilatation of the pupil and the intention tremor were controlled by this treatment. He did not doubt that the trouble was syphilitic, but could not accept the theory that all of the symptoms could be explained by one syphiloma.

Dr. Meierowitz replied that Dr. Francis Valk, in his recent ophthalmoscopic examination, had found the retina entirely normal. The absence of disturbance of speech and of nystagmus and the presence of papillitis seemed to him sufficient to exclude multiple sclerosis. It was, of course, quite possible that there were a number of lesions, but where the symptoms could be explained by one lesion, a diagnosis of a single lesion seemed the more rational.

#### TWO CASES OF TUMOR COMPRESSING THE CAUDA EQUINA; REMOVAL; RECOVERY.

Dr. B. Sachs reported these cases. He said that in both the symptoms had been relieved by operation. The first patient had been seen on September 3, 1897, in consultation with Dr. Wyeth. The second patient had been seen at the Mount Sinai Hospital in the service of Dr. Meyer. The first patient, fifty-six years of age, had been suffering for eighteen months with severe pains in the lower extremity and, more recently, with spasm of the muscles. He had been shot in the leg during the civil war. When seen by the speaker he had presented marked cachexia, severe pain and violent spasm. The pain was neuralgic in character, and radiated from the lumbar region of the spine down the posterior aspect of the thigh to the foot. The spinal column was not specially sensitive except over the second lumbar vertebra, at which point pressure elicited pains like those complained of ordinarily. There was a distinct diminution of tactile, pain and temperature sense at the upper and inner portion of the right thigh. The right knee-jerk was absent. The vesical and rectal reflexes were not impaired. The neoplasm was probably extradural, because, if in-



tradural, the symptoms would have been more symmetrical. Dr. Sachs had urged Dr. Wyeth to operate for the removal of the growth, and the operation had been done at once. Upon exposure of the spinal canal a tumor, the size of a small cherry, was revealed, adherent to the dura and bone. Microscopical examination of this mass showed it to be an alveolar sarcoma. As much as possible of the diseased tissue had been removed. Five days after operation the patient had been able to be up and around, and had been remarkably comfortable. The spasms had ceased. One month after operation the patient had been able to return to his home in the South.

The second patient was thirty-nine years of age, and had been admitted to the Mount Sinai Hospital on October 9, 1899. Some years ago he had been told that he had pulmonary tuberculosis, and had lived in California until apparently cured. The patient was of emotional temperament. He had come to the hospital with a diagnosis of locomotor ataxia. About one year ago his first symptoms had appeared. Examination had shown normal reaction of the pupils; no tremor of the tongue or face. There was a distinct kyphosis involving the twelfth dorsal and three upper lumbar vertebræ, but there were no tender points except between the third and fourth lumbar vertebræ. There was no marked interference with the vesical and rectal reflexes. The physical signs in the lungs were suggestive of tuberculosis. He had no strictly ataxic symptoms, but had distinct paresis of the lower extremities. The strictly unilateral character of the sensory changes pointed to a growth in the lower portion of the spinal column, but in deference to those who thought it possible that tuberculosis of the spine existed, suspension and fixation of the spine had been attempted. This had yielded an entirely negative result. The disease was evidently progressive, and mercurial and iodide treatment proved negative. On November 10, at Dr. Sachs' suggestion, Dr. Gerster performed laminectomy on the second and third lumbar vertebræ. At this operation a gelatinous mass was exposed, and the body of the third lumbar vertebra was found to be invaded by the disease. The tumor extended only to the lower margin of the second vertebra, and was removed. It had evidently compressed the cauda equina. Sections of the tumor showed it to be a small-cell sarcoma. Three days after operation sensation in the leg seemed to be improved. Improvement had been steady, but the patient had been compelled to remain in bed. There was reason to hope for at least a temporary recovery—certainly life had been prolonged by the operation.

Commenting upon these cases, Dr. Sachs said that the first

case had been diagnosticated as a chronic neuralgia previous to coming under his observation. The bilateral distribution of the pain, the absence of marked vesical and rectal symptoms, had seemed to him to point to the cauda equina, and not to the lumbar enlargement, and the tender spot had served as a valuable guide in both cases. There were few chronic spinal processes that followed the slow course of such spinal tumors. An exploratory laminectomy, if properly done, was practically harmless, particularly if done in the lumbar or dorsal regions.

Dr. Joseph Collins said that the cases presented should be a cause for much congratulation to the reader of the paper, as well as to the surgeon who operated. He felt sure that the mortality in these cases was not so great that neurologists should not be on the alert to diagnose them and urge operative intervention.

Dr. Peterson added his congratulations to those of the last speaker. It seemed to him that tumors in this particular locality were more difficult of diagnosis than in other portions of the cord. One important feature of the paper was the aid that the sensitive area or the deformity had given in establishing the diagnosis.

Dr. Sachs, in closing, emphasized the point that even if the sensory changes were very slight, they should be reckoned with in making the diagnosis. This had been well exemplified in the first case. Another point insisted upon was that he had managed by pressure upon a definite point to elicit the same pain as that of which the patient had complained. This had been very well marked in the first case.

#### DYSPHRENIA.

Dr. William Hirsch read a paper with this title. He said that the term "dysphrenia" had been applied to the secondary or sympathetic psychoses in contradistinction to the idiopathic or mental diseases, such as mania and melancholia. The secondary psychoses, which are produced by bodily diseases, are not characterized by the same uniformity of symptoms that mark the idiopathic variety. In the secondary psychoses there were frequent remissions with perfect lucidity during the course of the disease. Outbreaks of violence might be quickly followed by stupor. The speaker said that a further characteristic which he would call attention to was the occurrence of somatic symptoms, as loss of pupillary or patellar reflexes, rise of temperature, irregularity of the heart action and certain vasomotor phenomena, such as edema. In the secondary psychoses, the interstitial tissues, and particularly the blood vessels, were the ones at first and mainly affected. This was in accordance with the accepted pathology of the systemic spinal diseases. The changes in the interstitial tissues were produced by the diseases starting outside of the brain, such as the acute febrile diseases. There were a few cases in which, purely from the mental symptoms, one was justified in making a diagnosis of dysphrenia, even though ignorant of the exact nature of

the underlying bodily disease. A case of this kind was then reported by Dr. Hirsch.

The patient was a young and neurotic girl, seen by him first on September 5, 1896. She had then presented the condition of hallucinatory confusion. After an interval of quiet, on February 17, 1897, she became violent, and developed hallucinations of hearing and sight. At this time the temperature was normal. After about ten days she became stupid; her pupils were contracted, and the pulse was sixty. After about one week, automatic movements of the hands and head appeared. On March 14 menstruation came on, and she quickly became normal, and remained well for ten days. In April, 1897, she was given thyroid extract, and quickly recovered. She remained well for nearly two years. On February 2, 1899, she unexpectedly developed the same violent symptoms as before. She showed some transitory improvement again under the administration of thyroid extract, but soon passed into a condition of dementia. After about three months she became quieter; the pupils and patellar reflexes returned; the temperature became normal and her breasts, which had been much enlarged, returned to their natural size. Since that time her mental state had been good. The clinical features of this case evidently did not correspond to any primary psychosis. A loss of reflexes was generally considered as indicative of permanent change, but it was not impossible that this symptom might exist in functional disturbance. In the case just reported he believed that the menstrual disturbances were not the cause, but a symptom of the disease, as in the fourth, or worst attack, menstruation had had little or no effect on the mental state. The speaker suggested that the term "originary dysphrenia" should be applied to those cases, which, in their clinical aspects, resembled those known to be produced by toxic or infectious agents, but in which no cause for such infection could be found.

Dr. Brown said that he had seen a number of somewhat similar cases presenting apparently a physical basis for the mental disorder. In some cases of even very severe mental disease the mental symptoms would clear up during the later stages, for example, of a tuberculosis.

Dr. Mary Putnam-Jacobi asked Dr. Hirsch if he looked upon general paresis as a secondary psychosis, and also in what way the case of dysphrenia reported by him differed from recurrent attacks of hysterical insanity; also how far the failure to distinguish personality was really a mental symptom and how much the result of personal caprice.

Dr. B. Sachs said that probably all present had seen cases similar to the one described. He had himself had under observation a number of women between the ages of fifteen and twenty who had passed through very remarkable periodical mental changes. He had been much impressed with the suddenness with which these changes had

occurred. All of these patients had been members of strongly neuro-pathic families. They had passed quickly from a condition of mania to one of depression. A patient now under his care had regularly had periods of six months or more in which she had been in a condition of maniacal excitement, and had then very suddenly passed into an apparently normal condition, but really only a steppingstone to a period of excitement. It was questionable in his mind whether the class of cases spoken of in the paper represented a distinct entity in mental diseases. Such marked physical changes as described in the paper had not come under his observation.

Dr. Peterson expressed his belief that dysphrenia would be a convenient term for those cases that it would be difficult to describe under other names. The trouble was that these terms in psychopathy were usually founded upon clinical symptoms, with but little reference to the pathology, so that after a time the word came almost to include all insanity. Last summer, at Heidelberg, he had found about four varieties of insanity recognized, viz.: paresis, senile dementia, catatonia and dementia precox. About fifty per cent. of the cases in Germany at the present time were called catatonia, and the balance were included under the term dementia precox.

Dr. Hirsch closed the discussion. He said that it was certainly remarkable how many insane patients would pass through a disease like typhoid fever without developing any mental symptoms—indeed the patient whose history had been given had just passed through a typhoid fever in this way. He too recognized the great evil that had resulted from introducing names into psychiatry, but dysphrenia was not a new name, and certainly was useful in connection with a case like the one reported, which could not be well placed in any other classification. Dr. Sachs had referred particularly to circular insanity—to cases essentially chronic in their nature—but he had been discussing cases that were really acute. The mental disease consisted of a series of psychopathic conditions. The diagnosis of dysphrenia could only be made from a detailed history and long study. By a primary psychosis he meant a mental disease originating in the parenchyma of the organ; by a secondary psychosis one originating in the interstitial tissue; hence general paresis would be a secondary psychosis.

13. PARALYSE UND TABES BEI EHELEUTEN (General Paralysis and Tabes in Husband and Wife). Raecke (Monatsschrift für Psychiatrie und Neurologie, 6, 1899, p. 266).

Raecke has observed seven cases of paretic dementia in husband and wife, and in two of these syphilis positively occurred. There seem to be only 69 cases in the literature, omitting those of Crété and of Gottschalk recently published, and including Raecke's, of tabes or general paralysis in married couples, but probably the small number is due to imperfect observation. Syphilis was present 38 times in these 69 cases, and a history of probable infection was obtained in ten other cases. It was positively denied in two cases. General paralysis occurred in both husband and wife 27 times, general paralysis occurred in the man and tabes in the woman 14 times, tabes occurred in both 22 times, general paralysis in the woman and tabes in the man 6 times. It appears from these cases that the man is more liable to general paralysis than the woman, and that he shows the affection first, as demonstrated by 24 cases in which the husband first manifested disease and 9 cases in which the wife was first affected.

SPILLER.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Oct. 23, 1899.

The President, Dr. James Hendrie Lloyd, in the chair.

TWO CASES OF MUSCULAR DYSTROPHY (PSEUDO-MUSCULAR HYPERTROPHY) OCCURRING IN BROTHERS.

Dr. Charles S. Potts, presented two brothers, respectively 12 and 10 years. No similar disorder had been observed in the family. Neither boy walked until three years of age. No difficulty was experienced until the age of nine years, when weakness was first noticed in the legs. At present the older boy is unable to sit upright or stand. The movements of his arms, especially in elevation, are impaired. The muscles of his calves appear exceedingly large and unusually firm, and the infraspinal and deltoid muscles also appear to be well developed. The pectoral muscles and those of the back, thigh and upper arm are atrophied. The knee-jerks are absent. The muscles respond to the faradic current. The legs are cold to the touch and are cyanotic. A similar condition exists in the younger boy, but the disease is not so far advanced, and the boy is able to stand with some support.

A CASE OF INJURY OF THE SPINAL CORD WITH LOSS OF THE KNEE-JERK AND PRESENCE OF ANKLE-CLONUS,

FOLLOWED BY DISSOCIATION OF SENSATION OF HYSTERICAL ORIGIN IN NEARLY ONE-HALF THE BODY.

Dr. Charles S. Potts reported this case. The patient was 29 years of age. His family and previous history were unimportant. No evidence of any nervous disorder in the family or in the patient existed.

About two years ago, while trying to board a freight train, he fell and was struck in the back by the journal box. He at once completely lost power in both legs. He does not remember whether sensation was lost or not. When he was lifted up pain was felt in the back. In addition to the complete paraplegia, he was unable for four weeks to pass the urine except by using a catheter, but he knew when the bladder was full. From the end of this period he had incontinence for a space of several weeks. During this time the bowels were constipated, but if a purgative were given, he had no control of the sphincter. Priapism was also present. Two bedsores appeared over the sacrum and the legs wasted rapidly. In

about two months improvement began. He regained control of his sphincters, and in the course of six months could walk with the aid of a cane. About this time he noticed one day while washing his hands that the water, which was hot, felt hot to the left hand but not to the right, and he noticed also that the skin upon his right shoulder had been considerably abraded without his knowing it and without causing pain. He was admitted to the University Hospital in June, and at that time examination showed that the heart and lungs were normal. Marked scoliosis in the dorso-lumbar region of the spine was found, the point of greatest deformity being at the eleventh dorsal, the other vertebræ involved being the ninth, tenth and twelfth dorsal and first lumbar. He walked with the aid of a cane and with a marked steppage gait. All the usual movements of the legs could be performed but with some difficulty. He said that the legs were not so large as before the accident, but atrophy was only noticeable in the tibialis anticus and peroneal muscles. All the muscles responded to the faradic current excepting the muscles just mentioned, and these required a strong galvanic current and  $An.Cl.C.=K.Cl.C.$  The plantar, cremasteric and abdominal reflexes were present on the left side, but absent on the right. Both knee-jerks were absent and ankle-clonus was present. Both sphincters were well controlled.

Anesthesia to touch, pain and temperature was found on both legs in an area reaching from just below the knee to the proximal ends of the toes, and involving the great toe. The posterior aspects of the legs were not involved. Loss of pain and temperature sense and preservation of tactile sense were found on the right side of the body in an area involving the face below a line drawn from the junction of the coronal and sagittal sutures to a point about half way between the angle of the jaw and the point of the chin; involving also the right half of the neck, the right arm, and all the right half of the body and right thigh, excepting an area on the buttock extending about three inches from the middle line and an area varying from two to four inches wide extending down the thigh posteriorly. There was no anesthesia of the special senses or in the mucous membranes or skin of the penis. The right testicle was not sensitive to pressure, but the left was. No spots of hyperesthesia existed.

Examination two months later showed that the same conditions were present except that the legs had become stronger. A plantar reflex was obtained on the right side, the ankle-clonus was much weaker, and the areas of total anesthesia had become much smaller. Dr. Mellor found a reversal of the

color fields. Muscle sense was impaired in the right side, and the stereognostic sense was normal. When the plantar reflexes were examined the toes were flexed in the normal manner. There have not been any subjective nervous symptoms.

Dr. Francis X. Dercum said that the interpretation he would place upon this case was that the man had an actual spinal lesion due to trauma, and that in addition he had hysteria. The loss of thermal and pain sense which developed later, associated with reversal of the color fields, would suggest the hysterical nature. It is difficult to see how a lesion so low down could give rise to anesthesia extending so high up, even involving the face. A double lesion, however, was not excluded.

Dr. D. J. McCarthy reported a case of ambulatory automatism, probably of epileptic nature.

Dr. A. A. Eshner referred to a case which he had reported some years ago in a series of cases of hysteria in early life.\* It was that of a girl of 16 years, who came to Dr. S. Weir Mitchell's clinic. Her mother said that on several occasions the girl had under peculiar circumstances made long excursions—on one occasion so long as eighteen miles. On another occasion she misappropriated some money and went to Washington. She had had epileptic seizures. She was susceptible to hypnotism. While in her ordinary state she could not tell any of the stages in her trip to Washington, yet when hypnotized she could give many of the details. Dr. L. Witmer saw the patient in some of her hypnotic manifestations, and had the correctness of her statements tested. The father of this girl had some organic cerebral disturbance, the nature of which could not be ascertained; the mother was neurotic, and an aunt was a "mesmerist." The girl passed through the hands of a number of physicians. The last accounts of her were from reports in the newspapers. According to one account she was found under suspicious circumstances with a man in a disreputable neighborhood. On a later occasion the newspaper report gave an account of her suicide. Here, no doubt, there was a combination of hysteria with epilepsy in conjunction with ambulatory automatism.

Dr. William G. Spiller said that two or three years ago Dr. C. K. Mills and he had studied a case of ambulatory automatism in a young man who had epileptic convulsive attacks. He also had occasional intercurrent attacks of ambulatory automatism, in which he would try to leave the house, and on one occasion succeeded in doing so.

Dr. Francis X. Dercum remarked that ambulatory automatism associated with true epilepsy was not rare, but that in this case there were no other symptoms except the ambulatory attacks.

Dr. F. Savary Pearce reported the case of a man, aged 28 years, who had typical epileptic seizures, and during the last five years had had also attacks of ambulatory automatism. In the last year he had about three attacks. A peculiarity of the ambulatory condition is that it lasts a considerable length of time. He has been known to wander away from home and remain from one to six hours. During that time he is not aware of what he is doing, and is apparently in a condition of double consciousness. After the attack is over he is not able to report what he has done or said. He has been known to take the street car and pay his fare, and when he returned to his

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\*Pediatrics, August 15, 1897.

normal state was astounded at finding himself in some remote part of Philadelphia.

Dr. Wharton Sinkler said that a number of cases of the psychic equivalents of epilepsy without true convulsive seizures had been reported. Dr. Norman Henry had shown him a patient who had attacks similar to those in the case reported, but lasting a longer period; and in Dr. Henry's case no convulsive attacks had occurred.

Dr. D. J. McCarthy said that cases of epileptic automatism are not very rare, but the great majority of such attacks extend over hours, days, weeks, and sometimes months, and gradually merge into that condition known as double personality. The peculiarity of his case was the short duration, the attacks lasting only one to five minutes. It seemed more likely that these attacks should be truly epileptic than when they extend over a long time. There is always a question whether or not there is something more in these cases which may develop into double personality. In reports of the insane many accounts of cases are found in which the personality of the patient changes within a few weeks.

#### NOTE ON A CASE OF ACUTE POLIOMYELITIS

IN WHICH THE CEREBROSPINAL FLUID OBTAINED BY A QUINCKE PUNCTURE CONTAINED A DIPLOCOCCUS RESEMBLING THE DIPLOCOCCUS OF STERNBERG.

Dr. F. X. Dercum reported this case. The patient, a girl of two years, the fourth of five children of healthy parents, had always been a strong healthy baby. Her family history was negative as regards nervous or tuberculous affections. She had rubeola at one year of age, varicella at fifteen months and scarlet fever in April last, making a good recovery in each instance.

On September 2, 1899, while sitting on the second step leading to the porch of her home, she fell to the pavement. After a short "cry" she began to play, and the incident was forgotten. On the morning of September 9, the mother noticed that she tottered in walking, and that later in the day she was unable to stand. On putting her to bed she was found to have very little use of her arms and to be unable to hold up her head. The next morning, the paralysis was complete in arms, legs and neck. On the sixth or seventh day afterward a slight return of power in the left leg was noticed, and later in the right arm. Since then very little change has occurred.

At present there is no loss of sensation. The eye examination shows the pupillary reflexes to be normal, with no disease of the eyeground. Hearing is normal. There are no cardiac or pulmonary lesions. There seems to be no involvement of the mental faculties. There are alternate periods of irritability and quietude. She does not suffer pain and apparently has no headache. Constipation is marked. No involvement of sphincters. No retraction of head or abdomen.

At the request of Dr. Dercum, Dr. W. W. Keen made a



Quinke puncture; the fluid obtained was submitted to Professor William M. L. Coplin for examination, who reported as follows:

"Specimen consists of 2 cc. of clear fluid, watery in color.

"Microscopic examination in fresh state shows a few leucocytes and a small quantity of granular debris.

"Specimens stained by gentian violet and by Gram's method show numerous micrococci about .9 M. in diameter and arranged in pairs and extracellular; they stain by Gram's method, and morphologically and tinctorially they resemble the diplococcus of Sternberg.

"Inoculations on glycerin agar and blood serum failed to show any growth after incubation for 48 hours."

Dr. F. X. Dercum also reported a case of right-sided hemiplegia with hemianesthesia, right homonymous hemianopsia, jargon aphasia, Wernicke's pupillary reaction sign, and neuritic pain in the arm of the paralyzed side.

Dr. Joseph Sailer reported a case of sensory equivalent for the epileptic attack.

#### A CASE OF MIGRAINE WITH APHASIA, AND NUMBNESS IN ONE ARM.

Dr. S. McC. Hamill reported a case of migraine occurring in a young man aged 20 years. His father was a healthy, temperate man up to the time of the patient's birth. His mother, who died at about 40 years of age, apparently as the result of an apoplexy, was subject to violent attacks of migraine for a number of years. There exists a distinct lithemic inheritance on his mother's side. His health had been very good up to the age of 17 years. At this time he began to have mild attacks of hemicrania. In the beginning they were infrequent and unaccompanied by premonitory signs. After a few months' rest from his studies the headaches ceased, but as soon as his work was resumed they recurred again with greater severity at intervals of from four to six weeks, and were then for the first time preceded by the following phenomena, which have been present in all of his attacks up to the present time: He first experiences a visual disturbance which suggests a rotary scotoma; at almost the same instant he experiences a peculiar sensation in the epigastric region, which he has difficulty in describing, but which he says is superficial and relieved by removing the pressure of his clothing. Within a few minutes there develops numbness with loss of sensation; first in the little finger, passing in succession from the little finger to the thumb and rarely involving more than two fingers at a time. This sensation rapidly extends to the hand and up the forearm

to, but never beyond, the elbow. Usually following this, and only in some attacks, he experiences a sensation of numbness in his upper lip; one side of the nose (the side corresponding to that in which numbness in the forearm exists), and the entire tongue. On two or three occasions he has had difficulty with his speech. He has perfect knowledge of what he wishes to say, but is incapable of expressing himself. His head pain begins very shortly after these manifestations. It is always primarily unilateral, on the side of the head opposite to the side of the body in which the numbness exists. The pain in some attacks is extremely severe. It gradually disappears, and ordinarily terminates in from three to six hours. The premonitory phenomena usually persist from 20-minutes to a half-hour, disappearing gradually. Preceding and during the attacks the patient is quite restless, and apparently much frightened at the character of his symptoms. His health in the intervals between the attacks is fairly good. He has some disturbance of his digestion which is always exaggerated immediately preceding the attacks. There is no evidence whatever of mental deterioration, and there has never occurred anything which in any way resembled a convulsive seizure.

#### THE RELATION OF MIGRAINE TO EPILEPSY.

Dr. William G. Spiller reported two cases showing a relation between epilepsy and migraine, especially between epilepsy and the complex form of migraine.

The first patient had been referred by Dr. S. Solis-Cohen. He was a man fifty-one years old and had used alcohol since his eighth year and smoked to excess. He began when about forty-four years old to have numbness in the right side of the tongue and inability to speak. These attacks lasted a minute or two. The attacks occurred at first only once or twice a year, but are now frequent. When about forty-eight years old he began to have similar attacks of numbness in his right upper limb, and after a short time in his right lower limb; these attacks lasting a minute or two. The numbness of the hand was attended with weakness of the part. The paresthesia had always been confined to the right side of the body and had been associated with impaired function, as shown by loss of speech and the weakness of the hand. Consciousness was not lost during these attacks. The attacks in which unconsciousness occurred—only seven in all, and these within the last two years—began after the attacks of numbness had existed for some time, and they were preceded by an aura—a feeling of unusual good health followed by drowsiness. The bewilderment following an attack of unconsciousness, and a peculiar

cry preceding the attack, were quite characteristic of epilepsy. No convulsions had occurred, except in one attack within the last ten days, in which the upper limbs had become rigid for a few moments and the eyes were drawn upward. A slight tremor had been noticed a few times.

The second patient, a woman, was seen with Dr. S. M. Hamill. She suffered from attacks of paraphasia and unilateral headache with transitory paralysis and paresthesia on the side of the body opposite to the headache. The attacks began at the age of four and a half years after a severe fall upon the head. The case was one that would usually be called complex migraine.

Dr. Spiller referred to the literature on migraine and epilepsy. The severe forms of complex migraine may be regarded as "associated with" epilepsy, if one so pleases, but when epilepsy occurs with migraine it is well to recognize that there is probably some connection between the two diseases, and that it is not merely a chance occurrence of two affections in the same person. The important fact is, that a person who has a complex form of migraine, with temporary aphasia, paralysis and paresthesia, may some day have Jacksonian convulsive attacks. Whether this condition is to be regarded as epilepsy "associated with" migraine, or the whole is to be considered an epileptic manifestation, can be determined only after we understand better the pathology of the two diseases. It may be that complex migraine and epilepsy have the same cause in some cases, or it may be that migraine changes the structure of the brain and renders it liable to the epileptic disease. In some cases of epilepsy a history of migraine in the ancestors has been obtained.

Dr. Charles K. Mills said that his own tendency was to view true migraine and true epilepsy as distinct diseases, chiefly for the reasons presented in Dr. Spiller's paper. The non-occurrence of mental deterioration in cases of undoubted migraine is a very important point in differentiating the two diseases. Not only is migraine not usually associated with mental deterioration, but it is a disease very frequently present in persons of high mental attainments and great powers of cerebral resistance to outside impressions. He referred to a family in which migraine has been distinctly present in five generations. The presence of this disorder can be easily traced in two generations at present living. In this long line the mentality and general cerebral activity and strength have been good. As a matter of fact, we have epilepsy and epilepsy, if we choose to apply this term to some of the affections considered in this discussion, and to some other affections not mentioned. Of course, we cannot help recognizing the striking similarity in various particulars between migraine and epilepsy. He did not think that too much stress should be laid upon the argument in favor of the identity of the two diseases, because these disorders are, in some instances, associated; or even because, as in some re-

ported cases after the lapse of much time, motor paroxysms have appeared in the life history of a case of migraine.

Dr. Wharton Sinkler thought that there was a very close connection between epilepsy and migraine, but that it does not always follow that all types of migraine are associated with epilepsy. There are several types of migraine. One form depends on the uric acid diathesis, and there is another form in which the paroxysms seem to occur like those of epilepsy, without any appreciable cause. He thought that the relationship between epilepsy and migraine had been recognized for many years, and that a heredity of migraine in the epileptic is more frequent than a heredity of epilepsy. He believed that of the different forms, the ophthalmic type is more closely associated with epilepsy than the other forms.

Dr. F. S. Pearce said that an important point in the etiology was the presence or absence of indican in the urine. In both Dr. Sailer's and Dr. Hamill's cases there were well marked gastro-intestinal symptoms, and in the former indicanuria was noted. The question of auto-intoxication is to be borne in mind in the pathogenesis of some cases of epilepsy and migraine; and in the therapeutics of such cases, the gastro-intestinal tract should be carefully treated. The subject of indicanuria certainly has some weight in these cases in pointing toward toxic etiology.

Dr. James Hendrie Lloyd thought that there was no true ground for a confusion of diagnosis between migraine and epilepsy. The explosive neuroses, migraine, epilepsy and hysteria, undoubtedly have points in common, but he thought that they were not points of identity. Points of especial importance in the diagnosis are convulsion and loss of consciousness. There can be epileptic attacks without loss of consciousness and also without true convulsive attacks, but where there is loss of consciousness with a convulsive attack, there is an epileptic element. We do not have convulsive attacks or loss of consciousness in migraine. Exceptional cases have been reported, but he thought that the statement would hold good. The rare ophthalmoplegic migraine is rather a distinct species of migraine. It is not, as a rule, associated with convulsive attacks but with loss of power in some of the orbital muscles, which may last for days or weeks. A great error in nosology would be committed by classing all cases of migraine as sensory epilepsy. Migraine is different from epilepsy in its onset. It usually begins with some disturbance of vision. In some cases it begins with a sensory alteration in another part than the eye. In one of his patients the attack begins with numbness in a finger. This mounts gradually to the shoulder and disappears—the sensory aura not going up like a flash and ending in unconsciousness, but mounting slowly during fifteen or twenty minutes, and as it slowly passes away giving place to the characteristic headache. These points enable us to distinguish without difficulty between epilepsy and migraine.

Dr. Lloyd said that Dr. Spiller had found some cases, largely of this type of ophthalmoplegic migraine, where there seemed to be some epileptoid convulsions, but the speaker considered ophthalmoplegic migraine as of a special type or something more than ordinary migraine. Because in a few such cases there were slight epileptic phenomena, he thought that it did not give us a right to say that there was a distinct relationship between epilepsy and migraine, any more than between hysteria and epilepsy.

The fact that we may have the phenomena of epilepsy combined with those of migraine does not prove that the diseases are identical.

There can be the association of two neuroses in the same individual, as hysteria and epilepsy. This may be the explanation of some cases of epilepsy and migraine.

Dr. Francis X. Dercum was inclined to accept the view of the President, Dr. Lloyd, that these two diseases are separate entities, but that they do overlap in symptomatology, and that there may be infrequent cases in which it is difficult to say whether the case is one of migraine or of epilepsy. He believed also that they are diseases which in the main pursue an essentially different course.

Dr. Wharton Sinkler said that it was not so much a question of making the diagnosis between migraine and epilepsy as it was whether migraine resembles epilepsy and may coexist in the same patient, or if it is a forerunner of epilepsy. In his opinion the latter is not infrequently the case. In regard to the intellectual state of epileptics, it is well known that there have been a number of epileptics who have been very brilliant intellectually.

Dr. D. J. McCarthy thought that in these cases we lost sight of the reflex effect of pain in the production of a convulsive tendency. Irritation of the sciatic nerve will produce true epileptiform convulsions in the rabbit. He referred to the case of a man with a leg ulcer in the surgical ward of the Philadelphia Hospital who had had epilepsy when a child, but who had not had an attack for twenty or twenty-five years. One day the ulcer was curetted, causing intense pain with the production of a severe epileptic convulsion. Every time the ulcer was dressed there was such pain that a convulsion occurred. This case showed the reflex action of pain in the production of epileptiform convulsions. Where there is intense pain in an attack of migraine the reflex effect of pain in an unstable nervous constitution may explain the epileptiform attack.

Dr. William G. Spiller thought that there was some truth in the statement that gastro-intestinal disturbance may have some bearing on migraine and epilepsy. He considered the view of Dr. McCarthy, that pain may aid in the production of epilepsy, as plausible. It has been demonstrated that in long-continued neuralgia the blood vessels undergo change. Migraine may be due to a temporary vascular disturbance, and possibly may cause organic disease of the vessels, which in turn may lead to alteration of cerebral tissue. We know little of the pathology of migraine or epilepsy. That there is a clinical resemblance between the complex forms of migraine and epilepsy can hardly be doubted. Ordinary migraine does not as a rule, pass into epilepsy, and remains as simple migraine indefinitely; but temporary aphasia, temporary unilateral paralysis and paresthesia on the side opposite to the headache, seem to indicate that a grave disturbance is occurring in one cerebral hemisphere, and it is not surprising if symptoms of cortical irritation—convulsions—finally appear.

## Periscope.

### CLINICAL NEUROLOGY.

14. CHORÉE VARIABLE (Variable Chorea). E. Brissaud (La Presse médicale, Feb. 15, 1899, p. 73).

By this term is implied a spasmodic disease of the nervous system, differing from the chorea of Sydenham in that the movements are irregular, and involve different parts of the body at different times, and are sometimes associated with fibrillary contractions in the muscles. In many respects it resembles the habitual tics, or habit movements. Brissaud reports the case of a girl of 16, well nourished, but infantile in appearance. When admitted to the hospital, the symptoms were those of ordinary chorea, but the following day she developed abrupt movements of the arms and shoulders, the twitchings characteristic of chorea electrica, and from time to time profound sighing. The movements were exceedingly histrionic in character. At times she would remain perfectly quiet for periods of several hours. The first symptoms appeared at the age of 13½ years and lasted for four months. They then stopped suddenly and remained absent for a period of two months and re-commenced, each crisis resembling the preceding in character and duration, and the intervals of quiet seemed to be about equal. The father of the patient was an habitual drinker of absinthe to excess. The mother had fourteen children, seven of which died in infancy, and three of the remaining are suffering from tuberculosis. The patient presented various signs of degeneration. She was incapable of applying herself consecutively at any occupation for sufficient time to become expert, and is, therefore, obliged to do ordinary laboring work. The choreic movements while in the hospital were replaced by rough hawking sounds, and these in turn by coprolalia. This troubled the patient considerably; she made a vigorous effort to control it, and accomplished it, but soon developed a curious tic, whose symptom was cracking of the fingers. This disease is also known as polymorphous chorea (Magnan), and is one of the manifestations of degeneration that occur about the time of puberty.

SAILER.

15. HYDROCEPHALUS ACUTUS ACQUISITUS INTERNUS (IDIOPATHICUS) (Acquired, Internal, Acute Hydrocephalus). A. Heidenhain, (Berliner klin. Woch., 1899, No. 49).

Heidenhain reports four cases of idiopathic internal hydrocephalus. Three of these cases were confirmed by autopsy and one was cured by subcutaneous injection of morphine. The first case ran an acute course, although he would discourage any attempt to classify acquired hydrocephalus in adults into acute and chronic cases. A laborer, 40 years old, who was subjected to sudden variations in temperature at his work, was suddenly attacked with intense headaches, followed very rapidly by blindness and delirium. Temperature was normal, pulse slow, pupils were dilated and choked disc was present. The delirium was controlled by morphine hypodermically, and profuse perspiration established with sodium salicylate. The other three cases were intermitting in type and only one is described. A schoolteacher, twenty-three years of age, after a night of dancing and a ride of six hours in an open carriage, began the same day to have headaches. The head was bent forward on the chest, the shoulders drooped, the knees were

weak and bent, and when he attempted to walk he staggered considerably. He answered slowly and with difficulty and was able to follow questions with difficulty. After admission to the hospital wide variations in the pulse rate and temperature were noted, and later wild delirium. The autopsy in all three cases gave:—meninges anemic; intense anemia of the brain substance; the gyri were obliterated and the ventricles were widely dilated and filled with 350-500 grammes of a serous fluid. He concludes that trauma and exposure to cold are etiological factors, and the hydrocephalus is produced by a vasomotor reflex neurosis. The favorable termination of the first case he thinks was due to large doses of morphine, and that possibly subcutaneous injection of morphine or codeine, with hot douches and compression of the head, would be of service in the chronic cases. McCARTHY.

- 16 UEBER DAS LIDPHÄNOMEN DER PUPILLE. (GALAZZI) (Pupillary Reflexes). G. Mingazzini (Neur. Centralblatt, 18, 1899, p. 482).

The contraction of the pupils resulting upon forcible closure of the eyelid, which was described by Gifford and Westphal (*Med. d. Gegenw.*, 1899, No. 4), is declared by Mingazzini (*Neurol. Centralbl.*, 1899, No. 11), to have been observed as early as 1887 by Galazzi in Italy, and to have been described by him as a "lid reaction of the pupil." He declared that the phenomenon was due to a functional association of the contracting muscles, and pointed to the fact that closure of the lids was accompanied by a rotation of the eyeball inwards and upwards. The assumption that by the closing of the lids a compression of the bulb, and a resulting increase of intraocular pressure was produced, causing a contraction of the pupil through hyperemia, was rejected by Galazzi. JELLIFFE.

17. A CASE OF MYXEDEMA. Augustus A. Eshner (*International Medical Magazine*, 8, 1899, p. 822).

Eshner presents a case of myxedema in a man 50 years old, thick-set and short, a native of Russia. His family history is good. He complained first of pains in the epigastrium, the hypochondria, the lumbar region, the precordia and the muscles of the extremities. Food caused nausea and increased the pain in the epigastrium. His bowels were constipated and his appetite was good. Patient had a slight cough and expectorated a thick mucus, but his lungs were found to be unaffected. The tongue was large and filled his mouth, his lips were thick, the lower one everted, and his voice was hoarse. The hair was normal, though there were some areas of alopecia, probably due to a previous favus. In the axillæ the hair was scanty. The thyroid gland could be easily palpated. The patient could not close his hands, which were broad and spade-like, the right measuring 23.5 cm. and the left 22.5 cm. around the metacarpus. The heart was normal. Perspiration was abundant. Patient was always drowsy, though mentality seemed good. He was put on thyroid extract grs. v daily, and his lips, tongue and hands diminished in size, and became more mobile. He lost in seven weeks about eight pounds in weight. Treatment was irregular and patient finally disappeared from observation after the seven weeks of treatment.

Eshner thinks that there may be some relation between myxedema and the various conditions of morbid fat-deposition, or accumulation, as the thyroid may be atrophied in the latter, and they too are often benefited by the administration of thyroid extract.

Two photographs of the case accompany the article, together with one of a case of adiposis dolorosa, to show the difference between the two diseases. BONAR.

## Book Reviews.

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CLINICAL LECTURES ON NEURASTHENIA. By Thomas D. Savill, M.D.  
New York: William Wood & Co., 1899. Pp. 144.

To be at their best, clinical lectures should be heard, not read; the personality and magnetism of the teacher, the presence of the patient and the demonstrations of conditions, all help to impress facts and strengthen the enunciation of theories; but few such lectures can be crystallized in print and constitute a real addition to medical literature. The present five lectures are no exception to the rule. Although they have the great merits of being practical, earnest and reasonably brief, and of evidently having been written by an author interested in the work, and albeit they must have been of interest and value to the auditors for whom they were intended, they present nothing new, are necessarily unsystematic, and their rather numerous defects are decidedly more dangerous in cold print than they were in extemporaneous delivery. They are rather loose in conception, carelessly written, based on a questionable pathology, narrow in the treatment of a broad subject.

In the first lecture the author makes an admirable plea for clinical instruction in poorhouses—a plea which may well be addressed to the public and profession on this, as well as the other, side of the Atlantic. There can be no doubt that large infirmaries should not only be organized on a hospital basis, with adequate attending and resident medical staffs, but should be used in the preparation of physicians for their life work. The material in some of our larger cities is immense, and many diseases may be better studied here than anywhere else. Not only has the student the advantage of prolonged observation, but what is of paramount importance in many cases, the benefit of post-mortem examination. It is well to remember that Charcot made and maintained his reputation by means of the material in that great almshouse for women, the Salpêtrière.

PATRICK.

DIE NERVÖSEN KRANKHEITERSCHEINUNGEN DER LEPROA, MIT BESONDERER BERÜCKSICHTIGUNG IHRER DIFFERENTIAL-DIAGNOSE, von Dr. Max Laehr, Privatdocent an der Universität Berlin. Pp. xii-162. Berlin: Verlag von Georg Reimer, 1899.

The subject of leprosy, with particular reference to its manifestation in the nervous system, has received another excellent discussion at the hands of Dr. Max Laehr. Dr. Laehr was sent to study the disease in places where it was endemic, by the Medical Faculty of the University of Berlin. He chose the Balkan as the chief region for his study, and during a two months' journey made the careful investigations which form the basis of his monograph. The question is discussed largely from the clinical point of view, necessitated no doubt by the comparatively short period at his disposal for study, and the consequent difficulty of working out the details of pathological anatomy, even had an opportunity presented itself.

The always interesting and apparently still mooted matter of the relationship or possible identity of leprosy of the nervous system and syringomyelia, the author very rightly says must be settled by a consideration of the pathological anatomy, as well as the clinical course of the two affections. In general the feeling is certainly growing that



the diseases are not identical, and that leprosy has its origin as a disease of peripheral nerves, whereas syringomyelia is to be regarded as a primary affection of the cord. The question of secondary involvement of the cord due to extension from the periphery is a much more difficult problem to answer, particularly in view of our recent anatomical theories and the now well-recognized degenerations of the cord following or associated with so-called peripheral neuritis. The matter of central or peripheral origin of a process is clearly one difficult of determination, and it is apparently at this point that the progress of investigation in the leprosy question is now halting. The writer's material included a study of about forty cases, made at various places, of which numerous histories are given. The examinations were in all cases conducted in a systematic way, with particular reference to disturbances of sensation and of the functions of the cranial nerves. The examination of the motor sphere was confined to the determination of visible muscular atrophy. The results of these various examinations are given in characteristic detail, and will no doubt be valuable to the special student of the subject, who needs statistics and exact statement.

Following this section are nearly thirty pages devoted to a careful analysis of individual symptoms and their value in differential diagnosis. As a final outcome of his study Laehr believes that the important nervous disturbances of leprosy find their most natural explanation in a disease of the peripheral nerves, primarily their distal portions, but under certain circumstances in their more central segments, an opinion with which he finds the anatomical evidence (not his own) in complete agreement. In conclusion the author gives considerable space to an exhaustive differential diagnosis, for the most part arranged in parallel columns, between leprosy and syringomyelia, and leprosy and syphilitic polyneuritis. There are eleven pages of literature references appended, and a number of admirably reproduced photographs, illustrating superficial lesions. The entire study is clinical, rather than pathological, in a broad sense, a fact which lessens its value, particularly in view of the author's statement that some of the most important matters still in dispute demand a careful investigation of the pathological anatomy. There is, however, an implication that this further work will be forthcoming.

E. W. TAYLOR.

**THE CEREBROSPINAL FLUID. ITS SPONTANEOUS ESCAPE FROM THE NOSE. WITH OBSERVATIONS ON ITS POSITION AND FUNCTION IN THE HUMAN SUBJECT.** By St. Clair Thomson, M.D., F.R.C.S., Eng. Cassell & Co., London, 1899.

This admirable monograph is based upon the study of a single case, but Dr. Thomson has enlarged his paper by including the reports of all other cases hitherto recorded—only eight in fact—in which the same condition certainly existed; and by giving a complete account of the disease and the differential diagnosis from all affections with which it might be confused. Naturally, the most interesting part is the description of the condition of the patient observed by himself; a woman of 25, who at that time had suffered for  $2\frac{1}{2}$  years from a dripping from the left nostril. This dripping was almost continuous, and probably amounted to as much as 15 oz. in 24 hours. It was carefully analyzed by Prof. Halliburton, and found to conform in every respect to normal cerebrospinal fluid. It was absolutely sterile. The patient in youth had suffered from headaches, and on various occasions when the flow had ceased for a time, the headaches had reappeared. The nose and eyes were perfectly normal. There was no history of injury or other

satisfactory explanation for the condition. In addition to the eight cases collected from the literature, Thomson by thorough research has added 12 probable ones, and a number in which the condition possibly existed. All these groups are tabulated. He has also collected cases of various other conditions that simulate the escape of cerebrospinal fluid, particularly one of hydrorrhea, in which a chemical analysis was made of the secretion by Prof. Halliburton. The conditions can be distinguished by the fact that the cerebrospinal fluid contains no mucin, a trace of globulin, and pyrocatechin—the secretions from the nose contain pseudo-mucin, a considerable quantity of proteid matter, and a certain amount of sugar which reduces Fehling's solution. Among the most interesting facts recorded are the results of some experiments upon the effect of position and effort upon the secretion of the cerebrospinal fluid. It was found that when the patient was sitting quietly, the quantity was considerably less than when she made some vigorous effort, but that the increased quantities obtained during the period of effort were of lower specific gravity and contained fewer solids. The effect of position was also quite pronounced. When the patient was sitting up the percentage of solids was considerably greater. When the patient was allowed to sit in the chair and the abdomen was vigorously compressed, the quantity of fluid was almost twice as much as when she sat quietly. The percentage of solids was very much reduced.

We cannot refrain from expressing our satisfaction at finding a man willing to publish in the form of a monograph an article whose length makes it entirely too bulky for publication in a journal. The value of the book consists in the fact that it calls attention to a rare but interesting condition, and presents in succinct but complete form all our present knowledge upon the subject. It is, however, of more interest to the rhinologist than to the neurologist.

SAILER.

KRANKHEITEN DES RÜCKENMARKES. Prof. H. Obersteiner and Docent E. Redlich. Separat-Abdruck aus Handbuch der praktischen Medicin. unter Redaktion von Dr. W. Ebstein und Dr. J. Schwalbe; herausgegeben von W. Ebstein. Verlag von Ferdinand Enke, Stuttgart.

This chapter is devoted to diseases of the spinal cord and forms part of the "Handbuch der praktischen Medicin." It is 230 pages in length, and is quite profusely illustrated. It is intended more for the general practitioner than for the specialist in neurology, and will doubtless be of much practical value. The various diseases of the spinal cord are briefly but satisfactorily described. The names of the authors are a guarantee of the excellence of the work. Books of this character are always useful and this chapter could with advantage be published separately.

SPILLER.

ESSENTIALS OF THE PHYSICAL DIAGNOSIS OF THE THORAX. By Arthur M. Corwin, A.M., M.D. Third edition, revised and enlarged. Philadelphia: W. B. Saunders, 1899.

This excellent little book, which already is before us in its third edition, gives in a clear and concise form the elements of physical diagnosis. The systematic way in which the subject has been considered will make it especially useful for the student and will permit it to serve as a valuable guide to the more elaborate study of this branch of

medicine. It is gratifying to note that the plan so often followed by German authors, namely that of placing less important matter, disputed points and proffered theories in small print and not including them in the general text, has been pretty well followed. The section enumerating the physical signs of the separate morbid conditions has been retained and greatly enhances the value of the book. A number of schematic illustrations serve to admirably elucidate the text.

HENSEL.

MAKROSKOPISCHE UND MIKROSKOPISCHE ANATOMIE DES RÜCKENMARKS—  
MAKROSKOPISCHE UND MIKROSKOPISCHE ANATOMIE DES GEHIRNS.  
Prof. Theodore Ziehen of Jena. Gustav Fischer. Jena, 1899.

The work before us is the first of several on the anatomy of the nervous system, which together are to constitute the fourth volume of the comprehensive system of human anatomy now appearing from the publishing house of Gustav Fischer in Jena, under the editorship of Prof. von Bardeleben. Out of the 402 pages it contains, 341 are devoted to the macroscopic and microscopic anatomy of the cord alone, and it is needless to say that the subject is handled with the utmost minuteness of detail. In the remaining 61 pages the author gets only as far as the general consideration of the brain from a macroscopic standpoint, purposing to finish his task in the next *Abteilung*.

Prof. Ziehen has not confined himself strictly to human anatomy, but gives many interesting and valuable comparisons of the weights and the embryological, histological and morphological peculiarities of the human brain and cord and those of the lower orders. Like the recent work of Flatau and Jacobsohn—to which it is in certain ways similar—Ziehen's is not one which lends itself readily to analysis, but from the vast number of bibliographic references which it contains, it is obvious that nothing has been left undone to make the subject matter as comprehensive as possible. The illustrations, of which there are 94 in the text—some colored—are not, on the whole, in keeping with the rest of the work. The typographical errors are few, although this author, in common with many in this country, persists in putting an accent over the first e in Dejerine.

When completed, the work will certainly be of great value as a reference, both to the comparative anatomist and to the student of the minute anatomy of the nervous system.

J. W. COURTNEY.

ESSENTIALS OF ANATOMY. By Chas. B. Nancrede, M.D. Sixth edition, thoroughly revised by Fred J. Brockway, M.D. Philadelphia: W. B. Saunders, 1899.

While the use of quiz-compendes for students is not to be specially recommended, we have in this new edition of Nancrede, a book which is better adapted perhaps than most others for the use of those unable for lack of time or other reason to familiarize themselves with the more bulky text-books. The illustrations, so essential to the study of anatomy, are clear and comprehensive, but their number, we think, could have been increased without enlarging the size of the book, at the expense of histological details, since these, in most colleges at least, are studied from separate text-books. The anatomy of the abdomen has been entirely rewritten in accordance with the more modern description of Gray. The structure of the nervous system is not thoroughly handled.

HENSEL.

## SAUNDERS' AMERICAN YEAR-BOOK OF MEDICINE AND SURGERY.

The publisher has decided to issue this well-known work in two volumes this year, Vol. I treating of general medicine, Vol. II, of general surgery. Each volume will be complete in itself, and will be sold separately; each volume being about 600 pages in length. Publisher, W. B. Saunders, 925 Walnut street, Philadelphia.

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 MISCELLANY.

The fifth meeting of the Congress of American Physicians and Surgeons will be held in Washington, D. C., May 1, 2 and 3, 1900. President, Prof. Henry P. Bowditch, M.D., LL.D., D.Sc., Boston, Mass. Vice-presidents, ex-officio, The President of the American Neurological Association, Dr. Edward D. Fisher, New York City; The President of the American Gynecological Society, Dr. George J. Engelmann, Boston, Mass.; The President of the American Dermatological Association, Dr. Henry W. Stelwagon, Philadelphia, Pa.; The President of the American Laryngological Association, Dr. Samuel Johnston, Baltimore, Md.; The President of the American Surgical Association, Dr. Robert F. Weir, New York City; The President of the American Climatological Association, Dr. Abraham Jacobi, New York City; The President of the Association of American Physicians, Dr. Edward G. Janeway, New York City; The President of the American Association of Genito-Urinary Surgeons, Dr. James Bell, Montreal, Canada; The President of the American Orthopedic Association, Dr. Harry M. Sherman, San Francisco, Cal.; The President of the American Physiological Society, Russell H. Chittenden, Ph.D., New Haven, Conn.; The President of the Association of American Anatomists, Dr. Burt G. Wilder, Ithaca, New York; The President of the American Pediatric Society, Dr. Henry Koplik, New York City; The President of the American Ophthalmological Society, Dr. Oliver F. Wadsworth, Boston, Mass.; The President of the American Otological Society, Dr. Henry G. Miller, Providence, R. I. Chairman of the executive committee, Dr. Landon Carter Gray, New York City; Treasurer, Dr. Newton M. Shaffer, New York City; Secretary, Dr. William H. Carmalt, New Haven, Conn.

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THE  
**Journal**  
OF  
**Nervous and Mental Disease.**

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**Original Articles.**

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THE RELATION BETWEEN TRIGEMINAL  
NEURALGIAS AND MIGRAINE.

BY JAMES J. PUTNAM, M.D.,

PROFESSOR OF DISEASES OF THE NERVOUS SYSTEM, HARVARD MEDICAL  
COLLEGE.

In the year 1896 I published a series of cases illustrating an affection which I had studied for many years and which I designated provisionally as migranoid neuralgia of the fifth pair. The purpose of the essay was to call attention to the clinical peculiarities of the typical, recurrent form of supraorbital neuralgia, which differs widely from the neuralgias of the other branches of the trigeminal, and to emphasize its kinship to migraine, the fact being reverted to that Anstie used to consider migraine as a supraorbital neuralgia. This paper was published in the "Transactions of the Association of American Physicians,"<sup>1</sup> and in the *Boston Med. and Surg. Jr.*, Vol. 135, II, 1896. In the following year a brief but welcome critical notice of it was written by Möbius<sup>2</sup> in which the claim of a kinship between the neuralgia of the ophthalmic division and migraine was disputed, and the position was taken that a relationship between the two diseases existed only in so far as that both were indicative of a neuropathic tendency on the part of the patient.

There will always be those who take pleasure in drawing

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<sup>1</sup>"On Periodical Neuralgias of the Trigeminal Nerve and Their Relation to Migraine, with Special Reference to the Intermittent Supraorbital Neuralgia."

<sup>2</sup> Reference mislaid.

sharp distinctions between groups of phenomena, and those also who find satisfaction in noting the resemblances between them, and both of these methods, if honestly followed, have their value. The excellent work of Möbius has always been penetrated by the former of these tendencies, but sometimes to an extreme degree. It may be assumed as probable that there is some genetic relationship between the neuroses, analogous in a broad sense to that existing between the different animal species. It is not, in view of the best modern thought, to be assumed that, in either case, a distinct and complete series of gradations will ever be discovered. The ape is not the parent of the man, but the two organisms represent developments on analogous but slightly divergent lines. In the same sense, I do not claim that before the symptom complex which we call migraine (itself far from sharply defined) had taken its present form, it went through a stage now represented by the recurrent ophthalmic neuralgia. I reaffirm, however, that this affection stands out vividly from among the other neuralgias of the trigeminal nerve, and that some of the characteristics that it presents strikingly recall migraine, in such a way that the study of either disease throws some light on our knowledge of the other. I pointed out in the paper referred to the fact that not infrequently ophthalmic neuralgia occurs in families and individuals having a tendency to migraine, and that migraine of early years may turn later into ophthalmic neuralgia, and also that one sees occasionally, though very rarely, migranoid features characterizing the neuralgias of other branches of the fifth nerve.

I described a striking case of this sort which I had followed for a long time, where attacks of pain in the infraorbital area, which had seemed to originate in the disease of a canine tooth, assumed the migranoid type, recurring at intervals of several weeks, in the form of definite seizures, each lasting one day and being attended, as a rule, by nausea, and often vomiting, prostration, and sometimes hemicrania.

The object of the present communication is to reinforce this view by the report of another case in which an ophthalmic neuralgia of typical form, following, as it so often does, a sharp attack of coryza (usually coryza of the frontal sinus) and

recurring daily for several days at nine o'clock in the morning, to pass away again at one or two o'clock in the afternoon, was associated with temporary hemianopsia and other symptoms which are characteristic of migraine and certainly foreign to the clinical history of what we call neuralgias. I say what we call "neuralgias," because the names that we give and the lines that we draw, useful as they are, often serve to imply distinctions which are after all but artificial.

This patient was a young man of nineteen, a book-keeper, previously free from any tendency to migraine or neuralgia except that once he had pain over both eyes during an attack of coryza. This pain was not intermittent, so far as he could remember, and lasted but two or three days. His mother had been subject to typical migraine, but neither of his five brothers and sisters had suffered in the same way. During the few days previous to his visit to my office he had a sharp attack of coryza, which, however, was already better so far as the nasal discharge was concerned. On the second day before I saw him he woke in the morning with nausea and loss of appetite so that he was unable to eat any breakfast, in spite of which he went to his work as usual. That night he slept well. The next morning he woke feeling pretty well and took some breakfast and went to work. At about nine o'clock his manuscript began to look blurred, and a left hemianopsia rapidly developed, unattended by vibrating scotoma. After about five or ten minutes the visual defect passed away and instead of it a left frontal headache came on, accompanied with nausea. The headache continued until between one and two o'clock the same afternoon. There was no disorder of speech or paresthesia of the hand. In the afternoon he felt pretty well, but on the two following days the same occurrence was repeated, the third attack being very slight, perhaps on account of a large dose of quinine taken four hours before the time of the expected seizure. The temperature at the time of his first visit was 98 and his pulse 88. The hands were cold, as they had been off and on since the symptoms began. In other respects his physical condition was entirely normal. Ever since this attack which occurred two years ago, he has remained free from such seizures

The seat of the pain in this case coincided with the distribution of the ophthalmic division of the fifth pair, and in its duration and mode of recurrence the attack was distinctly like that of a typical ophthalmic neuralgia, and like it too in that it followed the stage of an acute coryza, while in other respects it was typical of migraine. On the other hand, it is certainly unusual for a person not subject to migraine to have a single attack for the first time at the age of nineteen and not again during the next two years and unusual too for migraine to disappear at noon-time and not to recur again until the next morning as late as nine o'clock, on several successive days, though histories of analogous sorts are occasionally given.<sup>3</sup>

The fact is that if we glance over the whole range of the typical painful affections of the different parts of the body—the head, the forehead, the face, the scapulo-brachial area, the intercostal area, the sternal and cardiac areas, the abdomen, the legs, the feet, etc.—we see that we have to do with a large series of disorders presenting more or less characteristically common features and likewise striking differences. Taking the character of the pain alone into account, there is but little, for example, in the sickening sense of localized intense pressure, so common in the intercostal region or at the lower angle of the scapula, to recall the rending pain of a sciatica, and still less in the “epileptiform” lightning flashes of a *tic douloureux* to recall the steady or throbbing ache of a supraorbital neuralgia.

In each of these cases the essential element of the disease is surely a dynamic or chemico-physical change, or peculiarity of structure, of some part of the central nervous system, and in each there is usually some evidence likewise of peripheral provocation of the outbreak.

What has been gained by putting the vast shifting collection of symptoms that we designate as migraine into a group by themselves? In spite of all that has been written as regards vasomotor theories is there any feature of “migraine” that justifies the assumption of a pathology different from that

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<sup>3</sup> See Möbius's monograph; Nothnagel's "Spec. Path. u. Therap." XII, iii, 1.



of the "neuralgias," or even any feature that does not have its analogue in one or another of these disorders? Certainly all of them, supraorbital neuralgia far more than the rest, present symptoms that indicate an involvement of nervous centers other than those whose functions are exclusively or directly sensory. Vasomotor changes, functional motor paralyses,<sup>4</sup> hysteroid symptoms, mental depression, attacks of "urina spastica" are liable to complicate them all. In one of the chronic "supraorbital" cases that was reported in my earlier paper a weakness and strange feeling in the legs preceded each outbreak of pain.

If the clinical history of migraine has to deal with a relatively large number of these accessory symptoms, and is in general more rich and varied, and if on the other hand the "neuralgias" are more often associated with pronounced neuritis, these differences are of degree and not of kind,<sup>5</sup> and are due partly to the fact that the centers involved in the case of migraine are of such high functional significance and united in such wide and close association with other cerebral areas.

In spite of the neuritis the typical neuralgias are diseases essentially of the nerve-centers, or at least of the nerve-centers in common with the nerve-trunks. The history of the peculiarly interesting, typically recurrent supraorbital neuralgia reinforces this view by furnishing an example of an intermediate condition, in the sense indicated at the beginning of this paper. In fact it is sometimes difficult and perhaps impossible to say, in a given case, whether one has to deal with an ophthalmic neuralgia or a migraine ("status hemicranicus" "hemicranica permanens sive protracta").

Such cases are the one described in this paper, others in the earlier paper, and many of those given in the classical treatises on migraine and in Mingazzini's paper

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<sup>4</sup> I have now under observation a young man who, in association with a continuous supraorbital neuralgia of several weeks' duration, has had a marked paresis of the oculomotor, and perhaps ciliary, branches of the third nerve, which gradually passed away as the neuralgia improved, both finally disappearing.

<sup>5</sup> It is well known that the supraorbital nerve often becomes very tender in migraine attacks, especially if of long duration. See also the closing references in this paper.

on the psychical complications of hemicrania.<sup>6</sup> The latter writer cites, for example, a case reported by Griesinger<sup>7</sup> where a patient, then forty years of age, had during the first years of life a simple right-sided neuralgia of the trigeminal which gradually passed over into what was considered a migraine, characterized by pain in the depth of the orbits and a sense as of a veil over the head, and also by the appearance of "visions" when the eyes were closed, consisting in strange figures, as of youths, of skeletons, etc.

In the same article reference is made to a study by Krafft-Ebing<sup>8</sup> of the psychopathic disturbances attending "neuralgias." In light cases this "dysphrenia neuralgica" consists only in irritability and melancholic depression, but in severe cases, "especially in neuralgia of the fifth and the intercostals, deep reaching disturbance of consciousness and even complete loss of consciousness may occur."

I will permit myself but one more remark, namely, that in spite of the large part that "neuritis" seems to play, and does play as a complication, in the clinical history of the neuralgias, not only the character but the distribution of the pain in these affections is mainly controlled by the "central" element in them and is to be studied as an example of "segmental" disorders in the sense of Head, and with his well-known observations in mind.

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18 BERI-BERI. Yamagiva (Virch. Arch., 156, 1899, p. 451).

This author declares that all the changes seen in the organs in beri-beri are due to purely degenerative causes and not inflammatory ones, and especially that it should not be considered as a form of polyneuritis. He states further that not a single parasite, so far as known, can be held accountable as a causative factor. His view holds that beri-beri is an intoxication due to the eating of certain kinds of rice at certain seasons of the year.

JELLIFFE.

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<sup>6</sup> Monatsschrift für Psych. u. Neurol. Feb., 1897.

<sup>7</sup> Not at hand in the original.

<sup>8</sup> In Maschka, "Medic. legale," Vol. IV, capit. "Follia alcoolica" S. 719.

# LESIONS OF THE OPTIC CHIASM, WITH A CLINICAL REPORT OF THREE CASES.\*

BY WILLIAM M. LESZYNSKY, M.D.

CONSULTING NEUROLOGIST TO THE MANHATTAN EYE AND EAR HOSPITAL,  
NEW YORK CITY, ETC.

These three patients with hemianopsia are presented in clinical illustration of lesions involving the optic chiasm.

*Case I. Partial blindness with atrophy of both optic nerves. Bitemporal hemianopsia. No other symptoms of cerebral disease. Hemiotic pupillary inaction in the left eye.*

H. K., born in the United States, 38 years of age, floor-walker in a drygoods store, was referred to me by Dr. P. A. Callan, October 14, 1899. He has been married 14 years, and has two healthy children. His wife has never aborted. During the last 8 years he has had two attacks of biliary colic. The last attack occurred 4 years ago. In both, gall-stones were passed through the intestine. One year ago he first noticed dimness of light at night. Last May there was difficulty in reading. Since then his vision has been failing rapidly. No headache; no diplopia. Bowels are regular, appetite good, and he sleeps well. No history of rheumatism, alcoholism or injury to the head. He denies syphilitic infection. He is gaining in weight. His wife says there is no change in his mental condition and that his memory is excellent. Family history unimportant.

The right pupil is larger than the left. Right = 5 mm. Left = 4 mm. Both react normally to light, and in convergence. Consensual reaction is feeble. Vision R. E. = 20-200 L. = 20-100. No central color scotoma. There is bitemporal hemianopsia for white, form, and colors; the vertical line passing outside and around the fixation point. In each temporal field for white, there are several islets in which perception is intact. There is some contraction of the remaining nasal fields both for form and color, this being more pronounced in the right eye, as illustrated in the accompanying chart.

Hemiotic pupillary inaction is present in the left eye. The ophthalmoscope shows optic atrophy on both sides.

Gait and station are normal. All reflexes are present and are normal. No objective sensory disturbance. Senses of smell and taste are unimpaired. No evidence of acromegaly.

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\*Read at a meeting of the Section on Ophthalmology of the New York Academy of Medicine, January 15, 1900.

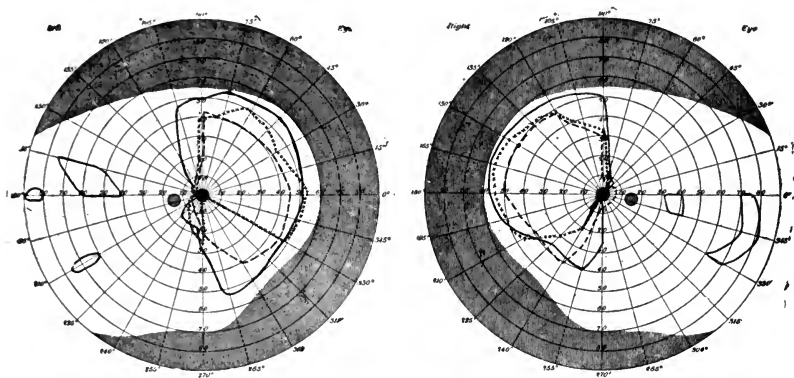
Further examination as to signs of disease of the nervous system or somatic indications of syphilis proves absolutely negative. Analysis of urine reveals nothing abnormal.

*Diagnosis.*—Lesion of the optic chiasm destroying both fasciculi cruciati.

Treatment with iodide of potassium, galvanism, and strychnine subcutaneously have all failed to make any improvement.

At the last examination a few days ago the visual acuity and the fields have diminished, as shown in the charts.

His general health, however, is perfect.



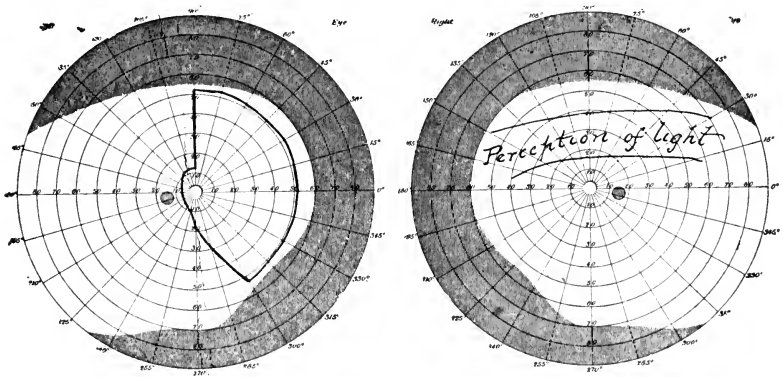
Case I. Lesion limited to optic chiasm, Jan., 1900.

——— field for white. \*\*\*\*\* field for red. - - - - field for green.

*Case II. Loss of vision in right eye. Temporal hemianopsia with left eye. Atrophy of both optic nerves. No other symptoms of cerebral lesion.*

J. S., born in Russia, 28 years of age, sewing machine operator, was sent to me by Dr. Cole of this city, September 22, 1898. He was always well until two years ago, when he was shocked at seeing a child run over and killed. For some weeks afterwards he remained nervous and apprehensive. Two weeks after witnessing this accident his vision began to fail, and now the right eye is blind. About a year ago he first noticed that he could not see toward the right with the right eye, and about two months later, the left eye was similarly affected, *i. e.*, he could not see toward the left without turning his head. About four months after the "fright" on

awaking in the morning after a good night's sleep, he was unable to raise his head on account of severe vertigo. This was increased by the slightest movement of the head. There was neither vomiting nor headache. He was confined to the bed two days. On the third day he was quite well again and returned to work. Three weeks later he had another similar attack lasting two days. He has had two milder attacks since, the latter having occurred nine months ago. Last November (ten months ago) he had acute purulent otitis affecting both ears. The discharge ceased in about a week, and he has had no further trouble. He has never suffered



Case II. Lesion of optic chiasm, Sept., 1898.

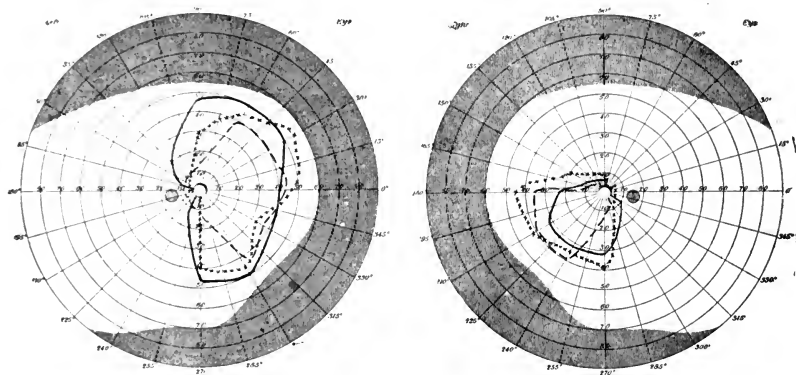
from headache, vomiting or diplopia. No injury of the head. No alcoholism or rheumatism. No history of syphilis. He has three healthy children and his wife has never aborted. He has been moderate sexually. Bowels are regular and appetite is good. He sleeps well. In fact, he says he is in perfect health save the trouble with his eyesight. Family history unimportant.

Pupils.—Right 4 mm. Left 4.5 mm. Right reacts feebly to direct illumination. Consensual reaction +. In convergence normal. Left, normal. Power of convergence is feeble; otherwise no muscular weakness. Vision, R = perception of light. Left = 20-100, with temporal hemianopsia, the vertical line of division passing around and outside of the point of fixation, part of the lower quadrant of remaining field being lost, as shown in the accompanying chart.

Ophthalmoscope reveals atrophy of both optic nerves. Sense of smell and sense of taste are normal. No motor or sensory disturbance. All reflexes are present and normal. Neurological examination otherwise absolutely negative. The urine shows nothing abnormal. Pulse 96, but regular. Heart and lungs present no signs of disease. No evidence of acromegaly.

*Diagnosis.*—Lesion of the optic chiasm destroying both fasciculi of the right optic nerve and the fasciculus cruciatus of the left eye.

He remained under my observation for three weeks, and then disappeared, but he returned to me a few weeks ago after



Case III. Lesion of optic chiasm associated with acromegaly, Oct., 1899.

— field for white. \* \* \* \* field for red. - - - field for green.

a year's absence. During this period he was under treatment at various ophthalmological and neurological clinics, receiving large doses of iodide of potassium, strychnine, etc. Now he is almost totally blind, being able only to indistinctly discern shadows and occasionally count fingers at about two feet. He has never complained of headache, and his general health is perfect.

*Case III. Acromegaly. Partial blindness of right eye, with atrophy of optic nerve. Bitemporal hemianopsia, plus the loss of superior nasal quadrant of right field.*

Mrs. K. W., born in the United States, 40 years of age, widow, was referred to me by Dr. N. J. Hepburn, October 12, 1899. Five years ago she had the grippe, the attack last-

ing three weeks. The symptoms were severe general headache, fever and pains in the extremities. She then noticed for the first time that vision was failing, and for awhile she was unable to read or sew on account of diplopia and vertigo. During the last three years she has suffered almost continuously from headache, which extends to the back of the neck. It is worse in the morning, but diminishes toward afternoon. Vision has gradually become worse until the right eye is nearly blind. Three years ago she noticed that she could not see toward either temporal side without turning her head. She now complains of headache and vertigo. She has never been pregnant. Menses are regular. Menorrhagia lasting eight days, and frequent leucorrhea. Always constipated unless she resorts to drugs. Flatulent dyspepsia. She also complains of occasional darting pains in the arms and legs, and edema of the feet and ankles. Formerly she was a very light sleeper. Now she sleeps soundly, and there is some somnolence during the day. Her face, hands and feet have grown larger. Three years ago she wore gloves size  $6\frac{1}{2}$ ; now she requires size  $7\frac{1}{2}$ . Formerly she wore shoes number 5; now she wears number 6. The hands and finger-joints are stiff and numb in the morning. She perspires readily. Her memory is somewhat poorer than a few years ago. During the last two years she passes a large quantity of urine daily. She is the youngest of nine sisters and three brothers. Three sisters, aged 14, 28 and 38 years, respectively, and one brother at 40 died of pulmonary tuberculosis. One sister, aged 30, died of renal disease, and another sister died during infancy. Two brothers and three sisters are living. The men in the family are healthy. One sister has pulmonary tuberculosis, another has chronic rheumatism, and the other has some eye trouble. Her mother died at 40 of paralysis. Father died at 86 of pneumonia.

Pupils equal at 3.5 mm. Right reacts feebly to light. Consensual +. Convergence —. Left, normal to light. Consensual and Convergence —. Vision, R. E. = 7-200. L. E. = 20-20 +. There is bitemporal hemianopsia for form and colors. In the *left* eye, the remaining portion of the field is concentrically contracted. In the *right* eye, only the inferior nasal quadrant is preserved, and that is contracted, as shown in accompanying chart. (See Chart III.) Ophthalmoscope shows right optic atrophy. She presents a clear clinical picture of acromegaly, as seen in the formation of the face, and the enlargement of the tongue, hands and feet, etc. Senses of smell and taste are normal. Thyroid not enlarged. Pulse 84 to 96, and weak. Heart's action

normal, save accentuation of aortic second sound. Lungs normal. No tremor. Muscular power in extremities good. Knee-jerks and other reflexes normal. Examination of urine negative.

*Diagnosis.*—Acromegaly, with lesion of the optic chiasm from pressure of enlarged pre-hypophysis, destroying both fasciculi cruciati and the superior half of the fasciculus lateralis of the right eye.

In another case of acromegaly recently reported by the writer<sup>1</sup>, the patient having been shown before this society last March, bitemporal hemianopsia was also present as a result of pressure on the chiasm by the hypertrophied pre-hypophysis. This patient died during the summer, but there was no autopsy.

Since the publication in 1881 of Wilbrand's classical monograph on hemianopsia there has been comparatively little advance in our knowledge regarding the subject of chiasm lesions, with the exception of Marie's description of acromegaly in 1886, and the incidental elaboration of the eye-symptoms in connection with the associated involvement of the pituitary gland.

Wilbrand collected and recorded 56 cases of chiasm-lesion with autopsy. This interesting report clearly demonstrates that disease of the chiasm is most frequently secondary, being dependent upon tumor originating in the adjacent intracranial structures. In 45 cases the tumor was located as follows:

|                                   |    |
|-----------------------------------|----|
| Sella turcica .....               | 15 |
| Hypophysis cerebri .....          | 11 |
| In the chiasm.....                | 6  |
| Floor of the third ventricle..... | 5  |
| Crista galli .....                | 4  |
| Base of skull.....                | 3  |
| Posterior perforated space.....   | 1  |

In 11 cases:

|  |   |
|--|---|
| Dilatation of the third ventricle from internal hydrocephalus .....          | 3 |
| Gumma of chiasm.....   | 3 |
| Tubercle of chiasm.....  | 3 |
| Tubercular caries of base of skull.....                                      | 1 |
| Extension of proliferation from a tumor of the optic nerve in the orbit..... | 1 |

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<sup>1</sup> Phila. Med. Journal, Oct. 7, 1899.



Four additional cases were each due respectively to periostitis, partial meningitis, cysticercus, and aneurism of the internal carotid artery.

In all of these cases the collateral symptoms were unmistakably indicative of intracranial disease; either meningitis or tumor formation.

In 1894 Sell<sup>2</sup> collected and reported 81 additional cases with lesion of the optic chiasm published since Wilbrand's article. From an etiological standpoint, the general character of the cases in this series corresponds with those of Wilbrand. Twenty per cent. were due to syphilis.

When lesions involve the optic chiasm, the prognosis as to the duration of life and the preservation of vision, necessarily depends upon the extent and character of the pathological process. There is a class of cases, however, in which the disease seems to be limited to the chiasm, the condition being ascribed to a local meningitis or periostitis, or to a permanent and non-progressive exostosis. This circumscribed lesion may gradually cause complete blindness of both eyes without any other discoverable symptom. Such individuals have been known to remain blind, with no further discomfort or additional manifestation attributable to cerebral disease.

I have shown two patients of this kind here to-night, and the late Dr. E. C. Seguin<sup>3</sup> published a report of three similar cases in 1887. I had the opportunity of examining two of them, and they presented all of the characteristics of this type. There was neither history nor evidence of syphilis or injury to the head, nor any disturbance of the general health.

It is well known that the interpeduncular space is the principal and favorite location of syphilitic basal meningitis; hence the frequent involvement of the third nerve and the other nerves to the eye-muscles, and also the optic nerves.

Oppenheim has shown that bitemporal hemianopsia may arise from such involvement of the middle portion of the chiasm. This hemianopsia may exist for a long time without any ophthalmoscopic changes. In 1886 he<sup>4</sup> reported a remark-

<sup>2</sup> Inaug. Dissert., Leipzig (Jahresbericht der Ophthal., 1894, v. 25, p. 174).

<sup>3</sup> Journ. NERV. & MENT. DIS., Vol. XIV.

<sup>4</sup> "Syph. Erkr. d. Gehirns," 1896.

able case of gummatous basal meningitis, with autopsy, in which the process had extended to the chiasm. There had been bitemporal hemianopsia, which had varied from time to time and had gradually disappeared, leaving a normal field. He thinks this changeability of the field is characteristic of syphilitic disease implicating the chiasm.

Since then several other cases have been reported. Such a possibility should always be borne in mind, for it becomes a very important element in prognosis.

It will thus be seen that chiasm lesions may be divided into four classes:

1. Associated with intracranial growths and their concomitant symptomatology.
2. From enlargement of the pre-hypophysis cerebri, as occurring in acromegaly.
3. In syphilitic basal meningitis.
4. From a circumscribed pathological process, which gradually produces complete atrophy of both optic nerves, without any cerebral symptoms whatever.

In the absence of evidence of papillitis in the cases of Class IV, it would seem that the nerve structure undergoes a slow degeneration due to gradual and persistent pressure, thus obliterating its conductivity, and this is probably the result of an adjacent local inflammatory process.

Of course, in the absence of post-mortem findings, it would be mere speculation to speak with any degree of definiteness as to the nature of the primary lesion in these cases. At any rate, whatever it be, it invariably terminates in progressive and permanent destruction of both optic nerves.

Without a careful perimetric examination of the visual fields the localization of lesions in the optic chiasm would be impossible.

## EPILEPTIC AMBULATORY AUTOMATISM.<sup>1</sup>

By D. J. MCCARTHY, M.D.

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The subject of epileptic equivalents, and especially that form of equivalent known as psychic epilepsy, has of late years received much attention from neurologists and psychiatrists. This condition has been variously described as psychic epilepsy, ambulatory automatism, ambulatory automatism comitial,<sup>2</sup> fugues,<sup>3</sup> dromomania.<sup>4</sup> The following case differs so essentially from those already described that I feel justified in recording it.

J. D., aged sixteen years, office boy by occupation, came to the Polyclinic Hospital complaining of "spells of unconsciousness." His father formerly used alcohol to excess, but otherwise the family history was negative. When two years of age the patient had "inward spasms." The body became rigid, the eyes rolled up, the head was retracted, and he remained in this condition a half hour. A year later the spasm was repeated and lasted several hours. For a year following this convulsion, he acted peculiarly, staring vacantly at times in front of him and refusing to answer questions. He enjoyed excellent health until he was ten years of age when he had several fainting attacks, usually in church, which his parents thought were due to the close atmosphere. About a year ago the present trouble developed. While going about doing his work, people would appear strange to him; should he be talking to anyone, anything they might say appeared peculiar and in a short time he would lose consciousness and walk rapidly away, or carefully scan the wall or the floor. In from two to five minutes consciousness would return and he would remember nothing of the occurrence. In one of the attacks the head was turned to the left, the eyes turned up and out. During the remainder of the day the eyes felt tired and sore. If questioned during the period of unconsciousness he would reply, giving his reasons for whatever he might be doing. In one of the spells he had a quantity of money with him and began to throw it away

<sup>1</sup> From the clinic of Dr. W. G. Spiller, Philadelphia Polyclinic. Read before the Philadelphia Neurological Society Oct. 23, 1899. (For discussion on this paper see p. 115).

<sup>2</sup> Charcot, "Lecons du Mardi," 1887-1888, p. 112.

<sup>3</sup> Charcot, "Lecons du Mardi," 1888-1889, p. 303.

<sup>4</sup> Régis, *Archiv de Neurologie*, 1894.

as he walked along the street. In one of the spells he knocked a glass of medicine from the table. When he regains consciousness there is no dull or heavy feeling or headache. The urine or feces have never been voided during an attack. A glass of water taken during the early part of the attack will prevent it. Muscular twitchings or jerkings have never been present. No hysterical symptoms have been observed. The examination of the patient shows a well-built young man of good color and without visceral disease. Mentality is good and active; he has no failure of memory, no hallucinations or delusions. The reflexes are normal, station is good and the gait is perfect. No disturbance of sensation is detected. Signs of injury to the scalp are not present.

The number of attacks during the present year by months are as follows: Jan., 2; Feb., 3; Mar., 3; Apr., 6; May, 4; June, 8; July, 10; Aug., 9; Sept., 8.

The nature of the disease upon which the ambulatory automatism presented in this case depends is rather obscure and worthy of a careful study. Ambulatory automatism, or that condition in which an individual consciously or unconsciously performs more or less complex ambulatory acts over which he has no control, is a symptom-complex occurring rather frequently in hysteria, less frequently in epilepsy, occasionally in neurasthenia, and rarely in alcoholism, degeneracy, etc. The ambulatory automatism of hysteria is closely allied to hysterical somnambulism and may be preceded by or follow an hysterical convulsion. It may and often does occur in patients who have never had an outspoken attack of hysteria, and in these cases stands in the same relation to the hysterical convulsion as does the epileptic equivalent to the epileptic convulsion. Charcot<sup>5</sup> would have us consider it an equivalent of the period of passionate attitudes, and refers especially to a noisy, turbulent form of hysterical automatism. It has certain characteristics which serve to differentiate it from the other forms of ambulatory automatism. The attack is rarely, if ever, of short duration and lasts usually hours, days, or even weeks. There are usually other stigmata of hysteria present, but sensory disturbances due to hysteria may be present in epileptic patients. The automatism alternates with, or may be immediately followed by, an hysterical convulsion. The pa-

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<sup>5</sup> *Loc cit.*, p. 326.

tient may remember what has occurred during the attack, or if there be a complete amnesia, it is possible to develop the entire history of the attack by hypnotizing the patient. This latter symptom has both a clinical and medico-legal value. Motet<sup>6</sup> presented to the Société Médico-psychologique a patient who had been arrested for entering a public urinal, soaking his handkerchief with water with the intention, he stated, of stopping a nasal hemorrhage. The police hesitated to accept the explanation of the experts of what they considered obscenity, until Motet hypnotized the prisoner and repeated exactly the scene. Pitres<sup>7</sup> also mentions a case in which valuable papers and jewelry lost during the fugue, were recovered by hypnotizing the patient and tracing the articles.

The ambulatory automatism of neurasthenia differs from that of hysteria, in that the patient is conscious of what he is doing, but impelled by a desire to wander from one place to another, is unable with his weakened will power and indecision to resist the morbid impulse.

Epileptic ambulatory automatism is that form of the affection usually described as psychic epilepsy. There are certain elements in the case reported above which led us to consider it epileptic in nature. The history of convulsions associated with unconsciousness in early life, the reappearance of psychological disturbances at puberty, the conjugate deviation in at least one of the attacks, the complete unconsciousness and the short duration of the ambulatory periods, in the absence of hysterical manifestations, point to an epileptic rather than an hysterical nature of the disease.

The occurrence of convulsions in infancy followed by psychological disturbances at puberty seems to us not unimportant in a diagnosis of epilepsy. We have lately seen at the Polyclinic Hospital an overworked girl of fifteen years, with an alcoholic heredity, who had from two to five epileptic convulsions daily for three years in early childhood. The convulsions entirely disappeared without treatment and she remained perfectly well up to the time of her first menstruation. Four days after the

<sup>6</sup> Mahet et Mesnet, quoted by Semelaigne, *Annal. méd-psych.*, 19, 1894.

<sup>7</sup> Pitres. *Ref. in Arch de Neurologie*, 1892.

first appearance of the menses, a severe epileptiform convulsion occurred, and since that time she has had several convulsions.

Gowers<sup>8</sup> also mentions a case very similar to this, of a boy who had isolated convulsions at three, five and seven years and at puberty true epilepsy with severe convulsions developed.

The short duration of the attacks never extending over a few minutes, and the complete unconsciousness during the period, also speak in favor of epilepsy. Attacks of such short duration rarely, if ever, occur in hysteria. Two cases at least are on record of an epileptic nature. Semelaigne<sup>9</sup> mentions the case of a man who in the midst of a conversation would suddenly lose consciousness, walk rapidly forward, then backwards and resume the conversation as if nothing had occurred. Mesnet<sup>10</sup> records the case of a judge who arose from his place in court, left the room and walked for about five minutes, returning to take up the work he so suddenly left.

The great majority of the cases of epileptic automatism reported have extended over a much longer period of time, but there is always a question in such cases of some mental disease complicating the epilepsy. In some of the cases reported, the clinical picture resembles that seen in the intoxications of various kinds.

The conjugate deviation noted in one of the attacks seems to be the only purely spasmodic muscular contraction in the course of the disease.

The ambulatory symptoms are not purely automatic and never spasmodic. They appear to be the result of a pathologic ideation after unconsciousness develops, or, to be more accurate, a retention of the faculty to carry on simple intellectual processes and to act on them during a pathological or abnormal condition of the consciousness. In all of the attacks in which the patient has been questioned he has given his reasons for his peculiar actions. He examined the sidewalk carefully in one attack because he "wanted a tape measure and thought it should be measured;" in another attack he stated that he

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<sup>8</sup> Gowers, "Dis. of the Nervous System," V, ii.

<sup>9</sup> Semelaigne, *Annal. méd.-psychologique*, 19, 1894, p. 71.

<sup>10</sup> Mesnet, *Archiv gen'l. de Méd.*, 1860, p. 147.

wanted "to go home," etc., etc. The answers are different in each attack and appear to bear no relation to any idea or conversation prior to the attack. There does not appear to be any fixed idea dominating his actions as frequently occurs in hysteria. In many respects the mental state in this and many of the cases of longer duration resembles that of the condition described as double personality.

The use of the bromides and bromalin in this case has not served to decrease the number of the attacks per month, but has shortened the period of the attack and prolonged the aura. As a result of this change he is often able, by securing a drink of water, to prevent an attack. In Charcot's case the attacks disappeared for a long time under the use of bromides and reappeared when the treatment was discontinued. Charcot considered this fact of much value and gave no other reason for his diagnosis of epileptic ambulatory automatism.

This condition together with the many other peculiar manifestations of masked epilepsy, described in recent years, indicates the difficulties in classification of epileptic conditions for practical clinical work. The somewhat arbitrary division of epilepsy into petit mal and grand mal, and the division of the latter into the aura, initial cry, tonic, clonic and post-convulsive phenomena of most text-books, while very valuable for teaching and demonstrating purposes, yet in the scientific investigation in our daily clinical work find rather a limited field of usefulness. The text-books tell us that petit mal is a condition of unconsciousness lasting a few minutes and at times associated with minor motor disturbances, and yet we find that Cabode<sup>11</sup> looks on unconsciousness lasting many hours and associated with complex automatism as an extended form of petit mal. The very small percentage of cases of grand mal having an initial cry, the relatively small proportion with a distinct aura, the variation in the presence and relation of the tonic and clonic stages, and the very wide variation of the post-convulsive phenomena and equivalents, all indicate the necessity of a detailed description of epileptic phenomena independent of the broad classification noted above. It is only through some of the minor manifestations of epilepsy that we

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<sup>11</sup> Cabode, Arch. clin. de Bordeaux, April, '95.

can expect to get the key to the pathogenesis of the disease. From a purely clinical standpoint we can divide epileptic manifestations into those purely psychical, purely motor, or purely sensory, or combinations of any or all of these. In his classification our case would be a psycho-motor form of epilepsy. All of the equivalents of epilepsy could be arranged according to this classification with the possible exception of the attacks of profuse perspiration which sometimes occur. If we accept the cortical control, or rather influence over blood pressure, as the experiments of v. Bechterew and others would seem to demonstrate, this profuse perspiration would be considered as a motor manifestation affecting the coats of the vessels.

In the discussion of ambulatory automatism in this paper I have left out of consideration the ambulatory automatism due to traumatism, alcohol, dipsomania, and degeneracy, because none of these factors were concerned in our case.

Many of the cases due to traumatism of the head are manifestations of a marked form of traumatic epilepsy, as Dewarze<sup>12</sup> has already pointed out. There are also cases where traumatic hysteria was present. Dipsomania as a manifestation of epilepsy and its association with ambulatory automatism has also been described,<sup>13</sup> but the relation may be considered as accidental rather than causative.

The ambulatory automatism of the insane has little interest in this connection, but the following case of automatism in a high-grade imbecile with epileptic attacks is interesting:

I. A., aged thirty-two years, was a high-grade imbecile at the Pennsylvania Training School for Feeble-Minded Children, who had epileptic convulsions once in three or four weeks. About a week before the convulsions occurred he had attacks in which he arose from his chair, dropped whatever might be in his hands and patrolled the rooms like a sentry, sometimes turning to the right. He got up from his chair at times and usually made one turn, seldom more. Often, a few days before he had his spasms, he got up at night, put on his clothes and wandered away from his room, and once escaped from the building. He was delusional at times. In one of the attacks he suddenly arose from his chair, turned once fully to the right, with a perceptible change in the color of the face,

<sup>12</sup> Dewarze, *Prog. méd.*, Nov. 16, 1895.

<sup>13</sup> Songues, *Archiv. de Neurologie*, 1892, t. xxiv.



and then he sat down saying, with forcible shaking of the right hand: "Don't do that." His urine showed a slight trace of albumin. I am indebted to Dr. C. K. Mills for the report of the above case.

I am also indebted to the kindness of Dr. Spiller in permitting me to use the material of the clinic for the preparation of the paper.

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19. REMOTE RESULTS IN ARTISAN'S PALSY. F. Savary Pearce (University Medical Magazine, 11, 1899, p. 578).

Pearce divides occupation neuroses into two great classes, the neurasthenic and the non-neurasthenic. The majority of cases are neurasthenic. The neurasthenic condition is the predisposing cause, the constant use of one member in a monotonous and limited movement is the exciting cause, and to these must be added the irritation of products of metabolism, formed in excess, or improperly eliminated, which is a factor that causes a really greater disturbance in the nervous system than is manifested in the affected part. The non-neurasthenic cases are more nearly of a purely local nature. These he subdivides into muscular, neuritic (neuritis or not), and vascular, or arterial. The muscular cases are exemplified in a brakeman, or a street car driver, in whom the constant using of the brake caused a soreness in the flexor muscles of the forearm. There was no tenderness of the nerves, or heat in the tissues. In the neuritic type there is a peripheral neuron irritability which may become a subacute neuritis. Telegraphers are of this class, and in most of them it is found that the ulnar nerve is not sufficiently protected by the tissues from pressure, or that they work with the arm in an improper position. A correction of their position, or the wearing of a pad about the elbow while at work, tends to prevent recurrence. Vascular cases are subdivided into neurasthenic and non-neurasthenic. The chief symptom of this type is paresthesia of the fingers and forearms, changing into all forms of delayed sensation, and cold and clammy hands. This condition occurs in young persons under thirty, and so can be easily differentiated from arteriosclerosis. An example of the neurasthenic type is a woman, overworked and anemic, who for years sewed upon a machine. The numbness in her fingers was caused by the constant irritation of the goods passing through them as she fed the machine.

The treatment for all kinds of occupation neuroses is rest, massage and galvanism, but appropriate treatment for the neurasthenic condition is essential. Pearce thinks that about one-half of all cases of artisan's palsy get nearly well, and about one-third of the other half get entirely well. An average of five years he considers to be about the time required for recovery in an average case of the neuritic type. In the muscular and vascular types recovery promptly follows the removal of the cause. In all neurasthenic cases, however, recovery is more slow. He cites a number of cases illustrating his classification, and the relation between artisan's palsy, neurasthenia, and uric acid diathesis, and the benefit to be derived from changes of climate.

BONAR.

## NEW YORK NEUROLOGICAL SOCIETY.

Annual Meeting, January 2, 1900.

### FACIAL AND RETROBULBAR NEURITIS.—PERIOSTITIS OF OPTIC CANAL.

Dr. William M. Leszynsky presented a woman, thirty-eight years of age, who had come to him first on October 11, 1898. At that time there had been a complete facial paralysis of the left side, which had existed for five weeks. There had been a loss of taste, but the eyes had remained normal. The facial paralysis had been preceded by severe pain and tinnitus in the left ear, lasting several days. She had been treated by galvanism in the usual manner. Two months later, while under treatment, she had complained of severe pain in the left supraorbital and left temporal regions, lasting continuously for over a week. She had then become totally blind in the left eye. The pupil was absolutely immovable, and the ophthalmoscope showed edema of the papilla, but nothing else. Within a week a well-marked papillitis could be detected in the left eye. The vision in the right eye was normal. Under treatment by mercurial inunctions and the internal use of iodide of potassium the vision had improved somewhat, and on November 4 the vision in the right had been 22-100ths. She now had optic atrophy, and the vision had decreased. The faradic irritability had returned, but was still below the normal. A diagnosis had been made of facial neuritis and retrobulbar neuritis arising from periostitis in the optic canal. This was a very unusual condition, and for this reason the patient was presented. Dr. de Schweinitz, of Philadelphia, had recently reported two or three cases, but in them the retrobulbar paralysis had developed nearly a year and a half after the neuritis. The case also showed that a return of faradic irritability did not necessarily mean a return of motility.

Dr. G. M. Hammond asked if uremia could be absolutely excluded in the case just presented.

Dr. Leszynsky replied that there had been a complete absence of all urinary signs and symptoms. The neuritis seemed to have been either of rheumatic or syphilitic origin. There were some elements in the case that had led him to think it possibly of syphilitic origin.

### SYRINGOMYELIA.

Dr. J. Collins presented a man whom he had seen with Dr. Powell. The latter had removed a small lipoma from the lumbar region, having no connection with the spinal cord or membranes, contrary to the opinion that had obtained before

operation. The patient was thirty-six years old, and had contracted syphilis nearly five years ago. Since that time he had been under almost constant treatment. He had complained of weakness in the right leg, pain in the back, and slight numbness in the left lower extremity. Careful inquiry had elicited the fact that while still a boy he had experienced difficulty in keeping the right leg in the stirrup when riding horseback, and had noted in that foot what had really been a clonus. The knee-jerk was exaggerated on the right side, and on that side there was also ankle-clonus. There was absolute thermoanesthesia from the fourth intercostal space on the left side down to the tip of the toe. The diagnosis was syringomyelia.

Dr. Richard H. Cunningham said that he remembered having tested the sensation of this patient some time ago, but he did not recall the existence of thermoanesthesia. He had always looked upon the case as one of tumor of the cord, and not as essentially syphilitic.

Dr. Pearce Bailey said that several similar cases had been met with at the Vanderbilt Clinic during the past three or four years. It was the custom there to examine regularly for such anesthesia, and while he did not recall the individual case, it was exceedingly probable if thermoanesthesia had been found it would have been noted and remembered.

Dr. L. Stieglitz was inclined to accept Dr. Collins' view of the pathology, although it was unusual to find such extreme spasticity in such an early stage. He would like to know if there was any muscular atrophy present anywhere in the body, and if there was any change in the sympathetic supply to the eyeball.

Dr. C. L. Dana also looked upon the case as one of syringomyelia. Some of the cases of slowly developing spastic paralysis without sensory symptoms seemed to him to be really examples of early multiple sclerosis. He had seen such cases develop characteristic eye symptoms after a good many years.

Dr. Graeme M. Hammond said he had had an opportunity of examining this man, and would agree entirely with the statement regarding the clinical symptoms. He would accept the diagnosis given by Dr. Collins. This case was certainly not one of recent development. He could not classify it except as one of syringomyelia.

Dr. Joseph Collins, in closing, said that the right lower extremity was one inch and a half smaller than the left. There were no ocular symptoms, and no vasomotor disturbances of the face. There might have been in the beginning an area of gliomatosis which, at first, was connected with the central canal.

#### TUMOR OF THE AQUEDUCT OF SYLVIIUS, OR OF THE CEREBELLUM; DOUBLE JOINTS.

Dr. Collins presented a boy of eighteen, exhibiting all of the cardinal symptoms of tumor of the aqueduct of Sylvius or of the cerebellum. About three years ago it had been first noticed that he was lethargic. Fourteen months ago, while climbing into a window, he had fallen, and shortly after this he had begun to vomit and to suffer from periodical headaches.

These had been followed by increasing sleepiness, and by a disturbance of the gait. His limbs could be thrown around, and the joints bent backward in a remarkable way. The kneejerks were feeble. There was no nystagmus, and no true cranial nerve or ocular palsies. Both eyes showed choked disk and slight pigmentation.

Dr. C. L. Dana thought this peculiar drowsy condition was characteristic of tumors in the aqueduct of Sylvius, extending forward into the third ventricle; yet, in other respects, the symptomatology was that of a cerebellar tumor. If the tumor were in the aqueduct of Sylvius it could not cause much distention or infiltration, or there would be some eye symptoms. The weight of the evidence seemed to favor a tumor in the cerebellum. It was possible that the double-jointed condition had been increased by his present affection.

#### A CASE OF HEMORRHAGIC ENCEPHALITIS AND MYELITIS.

Dr. Charles L. Dana and Dr. M. G. Schlapp presented this report, which was read by Dr. Dana. The subject was a man of sixty-seven years of age, who had come under observation last June. There was no clear history of intemperance. His previous health had been good. During the last seven years of his life he had been somewhat of an invalid. On June 4, 1899, he had complained of headache, and after taking two cups of tea, he had gone to bed. He had fallen out of bed a few hours later, and had then been discovered to have left hemiplegia. He had been removed to Bellevue Hospital. At that time his temperature had been 104° F., but the temperature had quickly fallen. On examination he could not protrude the tongue, and it was almost immobile. He was paralyzed almost completely on the left side. The lips were markedly affected. The abdomen was somewhat rigid. The patellar reflexes were normal, and ankle-clonus was absent. Sensation to heat and cold was normal. The urine showed nothing abnormal. Four days after admission he had had a chill and the temperature had risen to 105.5° F., and examination of the blood had shown the malarial plasmodium. After having been in the hospital for one week it had been noted that there was no paralysis of the right arm or leg, and the most notable feature was the paralysis of the tongue and lips. He was unable to make any distinct articulate sound. He could swallow and cough, and understood perfectly what had been said to him. Death had taken place on June 25.

At the autopsy made by Dr. Dana, only the brain and cord had been examined. There were slight edema and congestion of the arachnoid. The vessels of the base were not atheromatous. In the right hemisphere was an area of capillary hemorrhage and softening in the lower portion of the pos-

terior central convolution, and superior part of its marginal gyrus. In involved mainly the deep part of the upper lobe of the fissure of Sylvius. In the centrum ovale were two or three smaller areas of hemorrhage. There was no evidence of hemorrhage or softening in any other part of the cerebrum, cerebellum, or pons. The spinal cord showed two foci of softening, one in the upper dorsal and the other in the lower. The brain had been hardened in formalin, and then stained by various methods. The microscopical examination had shown in the affected area much dilatation of the blood vessels, small extravasations of blood, a great proliferation of cells, showing an irritative reaction to the hemorrhage—in other words, the evidence of encephalitis, apparently secondary to the disease of the blood vessels. There were also small areas of softening of the brain in the neighborhood of these hemorrhages, also the result of the breaking and obliteration of the vessels. The walls of the latter were somewhat thickened, but gave no evidence of a syphilitic process. Sections and drawings were exhibited. The examination of the rest of the brain had failed to show any evidence of sclerosis. The medulla showed no evidence of softening or of inflammation. Examination of the spinal cord had not yet been completed. The cord showed a small center of poliomyelitis in the dorsal region. Below this was a more extensive poliomyelitis, involving both the anterior and posterior horns. The appearances very closely resembled those described by other writers on encephalitis hemorrhagica.

Regarding the occurrence of speech disturbance in a case of this kind, Dr. Dana said that in the great majority of cases of this form of hemiplegia there was no speech disturbance except during a few days after the attack, which might easily be ascribed to shock. In another class of cases of hemiplegia there was a slight uncertainty in speech, lasting for several months. In still another class the hemiplegia was associated with some difficulty of speech, and sometimes true aphasia. He had never observed any sensory aphasia in these cases. When the lesion was well below the cortex, and involved the motor neuraxons and the collaterals going to the opposite hemisphere, there would be some hesitation in speech; when the lesion was still lower down, there would be no disturbance of speech at all. There was a very general opinion prevailing to the effect that pseudo-bulbar palsies of cerebral origin are due to lesions in the lenticular nucleus, but it seemed to him that our present knowledge of the anatomy of the brain made such a view untenable.

Concerning the disturbance of the tongue, the speaker said

that Dr. Collins, who had given a critical description of this subject, placed the tongue center in the anterior and upper part of the foot of the anterior central convolution. Some of the centers of the movement of the tongue were probably deep in the fissure of Sylvius, and not subject to experimental stimulation. In the case under discussion it was interesting to note that mastication and deglutition were not impaired. The case also brought up the subject of non-suppurative encephalitis. It seemed to be established that there was an infectious hemorrhagic encephalitis in infants; also that there was a hemorrhagic poliomyelitis in adults; also an acute hemorrhagic encephalitis of adults due to some acute infection, such as influenza. In the case presented there was a distinct history of malarial infection and of alcoholism, and this combination, it was reasonable to suppose, was responsible for the lesions discovered. Such a case might be classified under the name of degenerative hemorrhagic encephalitis. An examination of the autopsy records of sixty-seven cases of apoplexy showed that 42 of them were hemorrhagic, 11 embolic, and 11 thrombic, and 3 encephalitic.

Dr. E. D. Fisher agreed with the reader of the paper that, in a small percentage of cases, there might be encephalitis, and that the spinal cord might also be involved. He had seen cases affecting the right hemisphere in which the aphasia had been permanent. It was not common, of course, for such persons to lose the memory of names and things, but he could recall a few such cases. He did not believe there were enough fibers passing over from one side to the other to account for that. The right hemisphere he believed was the region in which the center of speech was fixed, and while it was more accentuated on the left, the right, in his opinion, could not be entirely excluded. This belief was based on his own clinical experience.

Dr. B. Onuf thought that, as a rule, dysarthria rarely occurred in connection with lesions of the internal capsule; it was more often met with in lesions of the cortical foci. According to his experience it was most frequently observed with lesions of the left hemisphere. To explain this he assumed that all the articulatory muscles were represented in each hemisphere, and that the center of the right hemisphere was subordinated to that of the left hemisphere through the fibers of the corpus callosum. If there was a lesion of the right hemisphere there might be a temporary dysarthria; if, however, there was a lesion of the left hemisphere the dysarthria would be permanent. On the other hand, if the lesion were in the internal capsule, whether on the right or on the left, dysarthria would occur. If the dysarthria were marked, it could be stated almost certainly that the lesion was in or near the cortex. He believed that there was a double speech center, and that that of the right hemisphere was subordinated to that of the left.

Dr. Fisher asked Dr. Dana whether he had noticed any difference in the character of the articulation in the first few days in a person with right hemiplegia and in one with left hemiplegia. Personally, he had found as much difficulty in the one case as in the other.

Dr. Collins thought the paper was most interesting because adequately proving that a single lesion was capable of producing diffuse

and multiple lesions in the central gray matter. This was the first communication of the kind ever presented in this country. The paper called attention to the possibility of an acute malarial infection giving rise to such a process. The changes discovered in this examination were those which might be expected theoretically. He did not see how this report could have much effect on the question of aphasia, because serial sections of the entire length of the medulla oblongata had not been presented—indeed, he was doubtful if the patient had had true aphasia. There might have been an affection of the central allocation of the tongue not implicating the muscles of the lips and palate, in part due to the very small focus of inflammation in the medulla oblongata. The explanation given by Dr. Onuff was the one that he had himself long held, and which was the same as that given by Dr. Dana in this report..

Dr. M. G. Schlapp said that the inflammation in the medulla seemed to be a very recent one. There was a small-round-cell infiltration, but the large proliferating cells found in the cortex were not present. The infiltrating stage and the proliferating stage were both represented in the cortex.

Dr. Dana, in replying to Dr. Fisher, stated that his experience had been that dysarthria occurred about as often with right hemiplegia as with left, and that dysarthria was practically due to the size of the lesion. If the lesion were very large and the hemiplegia complete there was almost always dysarthria. Where there was only slight hemiplegia associated with dysarthria it was probable that the lesion was high up in the cortex, or involved a part of it. He had not thought that this case illustrated aphasia, but rather dysarthria. The weight of evidence in this case seemed to indicate that a paralysis of the tongue was the result of a cortical lesion—a sudden apoplectic lesion. There had been absolutely no hemorrhagic process in the medulla.

#### REPORT OF AN UNUSUAL CASE OF LEAD PARALYSIS WITH AUTOPSY.

Dr. B. Onuf reported this case. The patient was a man, thirty-seven years of age, having a good family and personal history. He had been a painter for many years, and had had one attack of lead colic some years ago. A short time ago he had had a severe fall, and for some days afterward had appeared in a dazed state. From March until August he had done no painting; then he had begun painting again with colors containing a large percentage of lead. He had kept this up during the month of August, but during this time had suffered frequently and severely from colic. At the end of August he had become quite ill, and in three or four days had become almost helpless in the lower extremities and in the left arm. On admission to St. Catharine's Hospital in September, 1899, there had been complete flaccid paralysis of both lower extremities; absence of both knee-jerks; marked tenderness of the nerve trunks and muscles of the lower limbs. There was also paralysis of the flexor muscles of the left arm and of the extensors of the fingers. The gum showed a "lead line." Under diaphoretic treatment the spontaneous pain

had ceased. On October 4 he had had an attack of severe dyspnea, and on the following morning he died in a second and similar attack. The extensors and flexors of the right arm were found, at autopsy, wasted, as were also the peroneal muscles and the flexors of the thigh. The left lung was the seat of a broncho-pneumonia. Microscopical examination had shown what appeared to be poliomyelitis of the anterior horns. The parts examined so far had been the second and fifth lumbar, and there was an enormous infiltration of the walls of the blood vessels with round cells. This infiltration of cells had been so extensive as to lead to very general distention of the nerve cells. The anterior roots were markedly affected, but the posterior roots of the lumbar region were normal. The plantar nerve showed increase of connective tissue and endarteritis obliterans. The liver had been examined by Dr. Bookman for lead, but none had been found. As five weeks had elapsed such evidence was not of much importance.

Dr. L. Stieglitz said that this case showed in the human being exactly what he had found in his case of experimental lead poisoning. In experiments on 36 animals he had only obtained in one guinea-pig an acute poliomyelitis at the necropsy, and also cell infiltration and more or less destruction of the nerve cells around the foci. This guinea-pig had been paralyzed acutely. Death had occurred within twenty-four hours. In his experimental cases he had obtained, in every instance, changes in the ganglionic cells themselves. The case presented in the paper was, of course, an atypical one of lead poisoning, and hence would not justify any conclusions regarding the ordinary pathology of lead poisoning. The cases reported by Oppenheim and by Herter seemed to him the most typical.

Dr. F. D. Fisher thought the clinical history of this case corresponded more with that of alcohol than of lead poisoning. As a rule, in cases of lead poisoning the lower extremities were the parts first involved. He had written on this subject some years ago, and had called attention to the occurrence of these degenerative changes in the brain and cord in cases of alcoholism. It, therefore, seemed to him that the case reported combined both alcoholism and lead poisoning. The changes in the central nervous system had been pretty well established.

Dr. W. M. Leszynsky said that it also seemed to him that the reader of the paper had not proved the connection between the lead poisoning and the post-mortem findings. Certainly the case appeared to be one of alcoholic toxemia, or of some acute infectious process.

Dr. Hammond said that while it was very probable that the man had suffered from lead poisoning, he doubted very much if the lead had had anything to do with the condition of the spinal cord; it was much more probable that it was the result of some acute infectious process. He did not believe a case of true lead toxemia had been reported in which such changes in the nervous system had been observed.

Dr. Cunningham said that some weeks ago he had met with a case of lead palsy in a man who at the same time had developed a mild form of influenza. The phenomena observed had been quite similar to those reported in the paper.



Dr. Collins also took the ground that in addition to the lead poisoning there had been an alcoholic neuritis, or some acute infection giving rise to the changes in the spinal cord.

Dr. Onuf said that the frequent attacks of colic during the last few weeks that the man had been able to work seemed to show the close connection between the lead poisoning and the other symptoms. Cases of alcoholic neuritis usually present quite a different picture from that exhibited by his case. The autopsy had revealed evidence of a certain amount of alcoholism, it was true, but the man had not been drinking for a considerable time previously. There had been no history of an acute infectious process.

#### THE LATE DR. WILLIAM A. HAMMOND.

The New York Neurological Society desires to record its regret at the death of Dr. William A. Hammond, one of the founders of this society and for years one of its most active and distinguished members. Dr. Hammond was one of the pioneers of neurology in this country. His work was always suggestive and most lucidly presented, and it was often original and of permanent value. It has indissolubly linked his name with the history and growth of neurological science. It was before this society that many of his papers were presented. We record our appreciation of his work as well as of his brilliant mental gifts. These were most helpful to this society in its beginnings, and led to enduring results in the advancement of clinical neurology and psychiatry and the development of neurology in America.

CHARLES L. DANA,  
LONDON CARTER GRAY,  
GEORGE W. JACOBY.

- 
20. ZUR KENNTNISS DER SYPHILITISCHEN PSEUDOPARALYSE (Contribution to Syphilitic Pseudoparalysis). Oberwarth (Jahrbuch für Kinderheilkunde, 49, 1899, Heft 4).

Twelve cases of syphilitic pseudoparalysis of childhood are here reported. In four of the cases a careful electrical examination by Kalischer gave normal reactions. This fact, together with the rapid recovery, speaks against an involvement of the central nervous system, such as Zappert found in his case. In three of the cases some contractures or spastic symptoms were present. This was probably due to reflex spasm, caused by the great sensitiveness of the bones (periostitis). In most of the cases distinct evidence of periostitis was present, either in the presence of swelling or in tenderness along the course of the bones. This process gave rise to the rigidity of the muscles and the childish instinct keeping voluntary motion in abeyance. Evidence of organic disease of the nervous system; headache, vomiting, incontinence of urine or feces, or changes in the reflexes were not present. He draws the following conclusions: That pseudoparalysis syphilitica disappears in a few weeks under antisymphilitic treatment, leaving no evidence of organic nervous disease; that the clinical phenomena can be explained by an osteochondritis specifica; that the clinical picture is that of a more or less painful and complete paralysis of the extremities.

MCCARTHY.

THE PHILADELPHIA NEUROLOGICAL SOCIETY.

November 27, 1899.

The President, Dr. James Hendrie Lloyd, in the chair.

A CASE OF MYOPATHY LIMITED TO THE FACE.

Dr. F. X. Dercum presented a woman, aged twenty-two; Russian; unmarried, whose father, aged sixty years, and mother, aged sixty-eight years, were both living and well.



She had one brother and one sister who were also perfectly well.

The patient had always been in good health up to six or seven years ago. At that time a "swelling" made its appearance in the back of the neck. The swelling suppurated and was lanced. It was attended by a great deal of pain. About this time or shortly after, she noticed that her cheeks were becoming thin and this atrophy steadily and slowly continued until the present.

Her cheeks are now much flattened and concave, due to wasting of the facial muscles. The orbiculares palpebrarum are much wasted as are also the zygomatics and the buccinators. The lips are large and full; the orbicularis oris seems to have suffered less than the other muscles of expression. The angles of the mouth are quite full and slightly depressed. The muscles of mastication are not involved. When the patient smiles, the angles of the mouth are drawn upward, but not outward, or very slightly so. During rest the orbicularis oris as a whole drops somewhat forward, suggesting, in a slight degree, the "tapir-like" mouth observed in more advanced stages of the disease. When the patient is told to forcibly close the eyes, the resistance of the lids can be readily overcome. The wasting of the orbiculares palpebrarum makes the eyes appear unusually prominent.

Electrical changes other than a slight quantitative diminution were not present.

A careful general examination of the patient was entirely negative. There was no involvement of the scapular, shoulder or arm muscles, nor of the legs or trunk. The case is interesting in the fact that the affection is limited to the face, differing in this respect from cases of facial myopathy hitherto described. In all the reported cases there appears to have been an involvement of other muscles, especially the scapular and humeral muscles constituting the so-called facio-scapulo-humeral type of Landouzy and Dejerine.

Cutaneous sensibility, organs of special sense, knee-jerks and visceral functions were normal.

#### A CASE WITH TABETIC SYMPTOMS AND EXAGGERATED KNEE-JERKS.

Dr. W. G. Spiller presented a white woman of forty-two years, who had been referred to his clinic from Dr. Hansell's clinic by Dr. Reber. She had color-blindness and her vision was much impaired. Syphilitic infection was possible. Within the past year she had had severe shooting pains in the extremities. She had also had temporary ptosis and diplopia. She was easily fatigued, and when fatigued, staggered. Disturbance of micturition had existed for years. Slight ataxia was detected when the gait and station were carefully tested. Both knee-jerks were exaggerated, more especially the left. The pupils were unequal and Argyll-Robertson sign was present. Dr. Reber had found that she had incipient optic atrophy with contracted fields. During the past year she had had "giving way of the legs." Babinski's reflex was not obtained.

Dr. Spiller thought that this case was either one of tabes

or spinal syphilis, and that it might be an atypical form of the latter disease. The absence of the Babinski reflex indicated that the central motor tracts were not involved, and the exaggerated knee-jerks could not, therefore, be attributed to degeneration of these tracts if Babinski's sign were accepted. Optic atrophy was more characteristic of *tabes* than of syphilis. If the knee-jerks had been absent instead of exaggerated, the other symptoms were so marked that probably no one would have hesitated to call this a case of *tabes*.

Dr. F. X. Dercum said that he had had under observation a similar case in which there were pains, Argyll-Robertson pupil, well-preserved knee-jerks, and optic atrophy.

Dr. F. S. Pearce remarked that these cases were evidence of the fact that the sclerosis in its incipency may be limited to the upper portions of the cord rather than the lower.

#### MELANCHOLY AND HYPOCHONDRIASIS—CURED BY GYN- ECOLOGIC OPERATIVE SUGGESTION.

Dr. F. Savary Pearce reported the following case:

R. F., American, single, aged twenty-five years, reported to the Howard Hospital Nervous Clinic, October 25, 1898. She complained of an imaginary ailment—pregnancy—and was extremely hypochondriacal and melancholy as to the dreaded outcome of her state. A sadder picture of a delusion he had seldom seen. There was no history of hereditary nervous disease. She began to menstruate at fourteen years and suffered somewhat from the beginning with dysmenorrhea. Her mother died when the patient was twenty years old. This depressed her very much. Then she had added household duties, and this probably with a run-down condition added to the gradually increasing dysmenorrhea. In August, 1898, she imagined she had been raped and become pregnant, but it is almost certain that coitus never took place. The next menstrual period not coming until three days late, the woman was positive of pregnancy and had heard so much on the subject from an aunt that her salvation seemed to be the taking an oxytocic which she did in the shape of several cupfuls of a strong decoction of pennyroyal. This is said to have established the flow precipitously and with much pain. It should be mentioned that she had vomited the day before taking the drug and that this clinched the idea of pregnancy. Since then the periods have occurred about every three weeks. When she visited the clinic she was convinced that the drug had not been effective, and that she was still pregnant.

When examined on October 25, 1898, she was melancholic and hypochondriacal, extremely emaciated and delusional as to pregnancy. She gave no evidences of other insanity, the

one idea dominating all else was the rape and pregnancy. Tonics were employed for the building up of her depraved nervous system. A persistent acne and anemia were somewhat improved by Bland's pill and hygienic measures, but she could be induced to take but little food and her mental state remain unchanged. Finally on March 6, 1890, Dr. John B. Shober found the symptoms of obstructive dysmenorrhea. Pelvic examination showed acute ante flexion of uterus with elongation of supravaginal cervix—otherwise normal. Hymen absent. Dilatation of cervix and curettement of uterus were done under ether. The uterine sound entered four inches. A cure by this operation was promised.

The patient remained in the Gyncean Hospital several weeks under Dr. Shober's care, and when discharged she seemed more composed and her mental condition was greatly improved. On May 20, 1899, the note was entered that she was gaining flesh rapidly and had improved in every way, the delusion of pregnancy having remained away. Her menstrual periods had returned and were now practically normal, perhaps somewhat too frequent.

While there was slight gynecologic disease in this case sufficient to set up irritation, as shown by painful menstruation, still the cure of the mental depression seemed to be entirely attributable to the suggestion of doing away with the imaginary impregnation by the curettement.

#### EPILEPTIC AMBULATORY AUTOMATISM.

##### INTESTINAL AUTOINTOXICATION PROBABLE EXCITER OF SOME OF THE ATTACKS.

Dr. F. S. Pearce reported also the following case: A. F. G., aged 28 years, male, a very intelligent journalist, had enjoyed good health. He had always been a hard mental worker. Five years ago he had a very severe attack of septic typhoid fever with relapses; followed by post-typhoid meningitis. Convalescence was very long. For two years he was treated for persistent gastro-intestinal catarrh, followed a year later by marked gastrectasia and gastropnoia. Three years ago he had much improved in general vigor and somatic symptoms after a most rigid diet consisting largely of milk. His "stomach always remained sensitive," and from slight indiscretions in quality or excess of food he would suffer much for a fortnight from gastro-intestinal fermentation, depression, insomnia and palpitation of heart. He remained anemic. One day, from no apparent cause, he fell over after supper in a typical attack of grand mal. He has had half a dozen such attacks during the past three years, always preceded by especial exacerbation

of his chronic dyspepsia. During the past eighteen months he has had spells of acute indigestion, belching of wind, and tympanites. With these he first developed the epileptic phenomenon to be described, an attack of which was seen by Dr. Pearce on July 25, 1899, and which was as follows: The attack began by the patient calling for his wife. He was found running about the hall and into the bathroom in a dazed condition. He had the peculiar epileptic fixity of eyes and was much confused. He would talk of things that occurred during the day, finally stopping his wanderings to grasp tightly the bedstead, and belching a large amount of gas with some acrid stomach contents. He then was able to walk to his bed with assistance. He had no convulsion whatever, and from a dazed condition fell into a profound post-epileptic sleep from which he awoke in half an hour quite restored. A sea bath later in the afternoon caused him to belch gas freely.

The dilated stomach and evident state of intestinal fermentation in this man (indican was found in large quantities) led Dr. Pearce to the conclusion that primary intestinal irritation was the cause of the epileptic paroxysm, in a system predisposed to the epileptic state most likely by the post-typhoid meningitis referred to above. The sequence of indigestion from slight cause, discomfort in the epigastric region, borborygmus, and acrid eructations, has been too frequently seen to permit a doubt that such irritation does induce attacks at times as in the case recorded. The use of antifermentative treatment (salol) and the cutting down of the bromide of strontium to gr. x, *t. i. d.*, have much improved his general health, and the attacks have been less severe and considerably reduced in number.

#### A CASE OF SYPHILITIC PARAPLEGIA OF THE ARMS, THE LEGS ESCAPING.

Dr. F. X. Dercum presented a colored man, aged 29 years, who said that he had a chancroid (?) about four years ago. About seventeen months ago he was unable to move the right eyeball on account of paralysis of the muscles, or to raise the eyelid. He had no loss of vision, but suffered greatly from pain on the top of the head. In about three weeks both the paralysis of the eye and the headache disappeared.

About fourteen months ago the fingers of the right hand became weak and he could not properly open them or hold anything in them. Later the arm became weak and he could not raise it over his head. This condition came on gradually. About six months after the appearance of the symptoms on the right side the same condition occurred on the left, and

very soon, the patient states, he could not raise either of his arms above his head. The right hand slightly improved with time, while the left became somewhat worse. He occasionally had cramps in the muscles of his arms and fingers. He noticed that when the fingers were closed on the palms, he could not open them. The onset of the paralysis was accompanied by pain which was sharp and shooting in character, and worse at night. The pain seemed to begin in the neck and spread down the arms.

The headache persisted with varying intensity, and was also worse at night. For a time he was troubled with sleepiness, though for three weeks past he had suffered from insomnia.

There was no history of secondary syphilis. He did not have any internal treatment at any time.

At present the patient presents a striking and marked paralysis of both arms. He has paresis of both deltoids, and is unable to raise the arms beyond a right angle with the body. This is equally marked on both sides. He has also marked palsy of both triceps muscles and of the extensor groups of both forearms. In addition there is present marked wrist-drop in the left hand and almost complete wrist-drop of the right hand. He is also unable to extend the fingers; these assume the position of flexion due to the unopposed action of the flexors. The palsy of the extensors of fingers is most marked in the left hand, where it is complete. Neither the thumb nor any of the fingers can be in the least degree extended. In the right hand he has still slight power of moving the fingers and thumb in extension. Both biceps muscles, though small, are strong and show no evidences whatever of palsy.

In the arms there is a very pronounced wasting of both the flexor and extensor groups. In the left forearm the extensor group especially is markedly flattened. The supinator longus muscle, on the other hand, is well preserved, supination being performed readily and in an apparently normal manner. The interosseous muscles are decidedly wasted. There is no apparent wasting of the thenar and hypothenar eminences, although these muscles are exceedingly soft. In the right forearm there is likewise a flattening of the flexor and extensor groups, though the wasting of the flexor group is much less marked than in the left arm. Wasting of interosseous muscles in right hand, while evident, is relatively slight. The thenar and hypothenar eminences are normal. The supinator longus muscle as in the left arm is well preserved. Pronation and supination are performed with equal readiness in both forearms.

Electrical examination reveals marked quantitative diminution in the wasted muscles.

The movements of head and neck are performed freely in all directions. He states that there is a feeling of cramp or stiffness when bending the neck backwards.

There is no involvement of the legs. They are not paretic, nor is there any change in the gait. There is no involvement of the bladder, nor is there any history of such involvement. The tendon reactions are normal. This is true equally of the elbow-jerks and knee-jerks. The plantar reflexes also are normal. Sensation is well preserved to all forms. Pupils are unequal, the right being the larger; both react to light and in accommodation. There is a ptosis of the right lid; also paresis of the left externus.

The case is interesting because it departs so widely from the common form of spinal syphilis, the form which has been described by Erb. Cases of syphilis of the cervical cord without symptoms of lumbar involvement are extremely rare, and in the writer's experience the present case is unique. It is interesting further because of the almost symmetrical involvement of the arms; the paralysis is most marked in the same muscles and groups of muscles; for instance, in the triceps and extensors of the forearm upon both sides, while, on the other hand, the biceps and the long supinator upon both sides entirely escape. It is important further to note that the palsy to some extent approximates the form of brachial palsy known as the lower arm type, and this fact enables us to localize the lesion with considerable accuracy. The lesion must involve the cord slightly above or at the level of the seventh and eighth cervical and first dorsal roots. That it was not absolutely confined to these levels is proven by the partial involvement of the deltoids, though the remarkable preservation of the biceps and supinator longus shows that the lesions at the upper brachial levels cannot be pronounced.

Another point worthy of attention is the fact that the involvement of the arms, though symmetrical, is not equal in degree, one arm being more decidedly affected than the other; we have here an analogy with syphilis of the lumbar cord, the involvement being generally a little more pronounced upon one side than upon the other.

Note—Since the patient was presented to the society active specific treatment has been instituted, with the result of a very marked improvement in the paralysis. The patient is now able to perform all of the normal movements of the arms, while all of the paralyzed muscles show a decided return of strength. This observation is important as demonstrating the undoubted syphilitic nature of the case.



## ISOLATED ABDUCENS PALSY, PROBABLY PERIPHERAL.

Dr. Wendell Reber, by invitation, reported a case of isolated palsy of the left external rectus. A woman of 50 years came to the clinic of Dr. Hansell at the Polyclinic Hospital with paralysis of the left external rectus. Four weeks previously she awoke with rather obscure vision and more or less diplopia. These conditions became more pronounced the following day. No history of trauma or of any antecedent acute disorder except rheumatism could be obtained. Two brothers and two sisters of the patient were the subjects of a more or less aggravated lithemic middle ear deafness, and the patient herself was deaf. No nervous or orbital disease could be found to explain the abducens palsy. The woman slept with the head of her bed between two windows, and with much draught upon her. She also slept on her right side, so that the left side of her face was exposed. The case was thought to be one of palsy in a rheumatic person following exposure to draught. Considerable motion was regained in paralyzed muscle by the use of strychnine, the iodides, the salicylates, electricity, etc.

Dr. Reber said that little could be found in the literature concerning such cases, and quoted the opinions of several writers.

Dr. William G. Spiller said that he had been much interested in this case because of the possibility of the abducens palsy being due to rheumatism, or exposure to draught; as occurs in facial palsy. The patient slept with the affected side of the face exposed to a draught; she had had rheumatism two years ago, and at one time had facial palsy. About a year ago Dr. de Schweinitz read a paper before the society reporting two cases of retrobulbar neuritis in patients who had had facial palsy. A relation seemed to exist between the two conditions. Dr. Spiller thought that abducens palsy might be related to facial palsy in the same way, as he had a case under observation, sent to his clinic by Dr. Risley, in which abducens palsy followed facial palsy of the same side a day or two after the exposure to a draught.

A CASE OF AMYOTROPHIC LATERAL SCLEROSIS IN WHICH DEGENERATION WAS TRACED FROM THE CEREBRAL CORTEX TO THE MUSCLES.<sup>1</sup>

WITH REMARKS ON THE EXTENT OF THE CORTICAL MOTOR AREA IN MAN.

This was the title of a paper read in abstract by Dr. Spiller. Dr. Spiller said that the cases of amyotrophic lateral sclerosis with necropsy and microscopical examination are not very numerous, and those in which the degeneration of the central motor tracts has been traced throughout the cord and

<sup>1</sup>This paper will be published in full in the memorial volume to be issued from the William Pepper Laboratory of Clinical Medicine.

brain into the central cortex number only ten, and in only two of these (case of Probst and case of Spiller) has the corpus callosum been examined and found diseased.

In the case reported by Dr. Spiller the degenerated motor fibers were traced throughout the central nervous system from the Rolandic area to the sacral region, and degeneration was found in the corpus callosum, ulnar nerve, and the muscles of an upper limb. The degeneration of the cerebral cortex was employed in the determination of the extent of the cortical motor area in man.

The patient was in the service of Dr. C. K. Mills at the Philadelphia Hospital, and the clinical notes and pathological material were obtained from Dr. Mills. A brief abstract of the case was as follows:

A man, 55 years of age, a laborer, contracted a severe cold in October or November, 1897. He said he had been healthy until that time. Toward the end of 1897 he began to have dysphagia, involuntary dribbling of saliva and loss of power in the legs and arms. These symptoms increased until he became confined to his bed, and was almost completely paralyzed. The deep reflexes were exaggerated, and fibrillary tremors were noticed in the tongue and other muscles. Sensation was not disturbed. Muscular atrophy was intense. Death occurred November 17, 1898.

The disease began with bulbar symptoms and involvement of the limbs, and ran a rapid course, the patient dying about a year after his symptoms first became very perceptible.

The muscles of the upper extremity examined were atrophied, and those near the distal end of the limb were more altered than those near the proximal end. In an interosseous muscle examined the atrophy was found in an extreme degree, the muscular fibers were small, but many still preserved the transverse striation; some had only longitudinal striation. Fatty degeneration of muscular fibers could not be found. The interstitial connective tissue was increased in amount, and much interstitial fatty tissue was found. The intramuscular nerve fibers and the muscle spindles appeared to be normal.

The ulnar nerve when teased and stained with osmic acid was found to be partly degenerated. The changes in the ulnar nerve were comparatively slight.

The anterior roots of the spinal cord were much atrophied, and in some sclerotic areas were found.

The nerve cells of the anterior horns were greatly degenerated, especially in the cervical region, although those in the lumbar region were also much diseased.

The crossed pyramidal tracts and the right direct pyramidal tract were degenerated throughout the cord from the motor decussation into the lumbar region. The sclerosis of the crossed pyramidal tracts could be followed into the sacral region. Degeneration in the motor tracts could be traced by Marchi's method from the lumbar region to the motor cortex.

The hypoglossal nuclei and intramedullary portion of the hypoglossal nerves were altered. The posterior nucleus of the vagus contained some highly pigmented cells. The nucleus ambiguus contained few cells, and some of these were possibly diseased. The seventh nuclei were normal, except that many of the cells were much pigmented. The posterior longitudinal bundles were slightly altered.

Distinct degeneration of the fibers within the motor area of the cortex was found by Marchi's method, and the degeneration of the corpus callosum was intense. The cortical nerve cells within and without the motor areas were greatly pigmented, and black granules were found by Marchi's method in the area of the tangential fibers. The giant cells of the paracentral lobule were numerous, but much pigmented.

The cases of amyotrophic lateral sclerosis in which degeneration has been traced throughout the pyramidal tract to the cortex are two of Kojewnikoff, two of Charcot and Marie, one of Lennmalm, one of Lombroso, one of Hoche, one of Probst, one of Mott, and one of Spiller, numbering ten in all literature.

The degeneration of the corpus callosum was intense in Dr. Spiller's case. Distinct parallel rows of black dots could be seen all through the sections from the middle portions of the callosum. The connection of the motor areas of the brain as indicated by these findings must be a very intimate one.

The changes in the peripheral muscles and ulnar nerve in Dr. Spiller's case were interesting. Those muscles nearest the distal end of the upper limb were most wasted. The greatest alteration was found in the first interosseous, and the supinator longus was almost as much diseased, while the biceps and deltoid were not so much affected. Even in the first interosseous, normal muscular fibers were found in the midst of the intensely atrophied ones. In the supinator longus atrophied fibers were found, occurring in groups in close proximity to normal ones. This explains why reaction of degeneration may be absent in amyotrophic lateral sclerosis. The microscopical study shows that one muscular fiber after another undergoes almost complete atrophy, and that even late in the process normal muscular fibers are found in the most altered tissue.

In these highly atrophied muscles the intramuscular nerves were normal. There is no reason for the belief that atrophy of muscles of necessity causes alteration of intramuscular nerve fibers, detectable by the Weigert's hematoxylin method at least. Probably some of these intramuscular nerves were sensory.

The muscular fibers in sections taken from the right side of the tip of the tongue were much atrophied, but by Marchi's method did not contain any evidence of fat. Many muscular fibers were perfectly normal. In view of the distinct atrophy in the twelfth nuclei, and of the fibrillary tremors of the tongue seen during life, this alteration of the lingual muscles was expected. The interstitial fatty tissue was very abundant, and many of the muscular fibers were very small. The intramuscular nerve fibers of the tongue stained by Weigert's hematoxylin method appeared to be somewhat degenerated.

Amyotrophic lateral sclerosis is essentially a disease of the motor system, and it occurred to Dr. Spiller that the degeneration of the cerebral cortex in his case could be employed in determining the extent of the cortical motor area in man. This has never been done in any of the few cases of amyotrophic lateral sclerosis with cortical degeneration reported in the literature.

Our knowledge of the extent of the motor cortex in man is very incomplete. Lemaq has attempted to define the motor area by the results of the electrical irritation of the human brain at the time of operation, and he has depended very largely on the work of American investigators (Keen, Mills, Starr, Dana, Lloyd, and others). The diagram of the motor area determined in this manner is imperfect.

Dr. Charles K. Mills said that the paper read by Dr. Spiller was one of the most valuable contributions ever presented to the society. It demonstrated the extent of the motor cortex in man by an entirely original method, and the pathological findings in general were of unusual interest and importance. It seemed to him that Dr. Spiller's results, in so far as they fixed the limits of the motor cortex, were a further confirmation of the views which the speaker had long held and taught, viz., that the motor region is in all essential respects separate from the sensory region of the cortex. This motor area he believed is limited, as shown in his published diagrams, almost exactly as determined by Dr. Spiller. Dr. Mills believed that the central convolutions and the parts corresponding to them on the median surface of the hemispheres, and the posterior extremities of the first and second, and, to a certain extent, of the third frontal convolution, together constitute the cortical motor area. He also still held that the great sensory area of the cortex (the region of representation of muscular and cutaneous sensibility), is composed of the superior parietal lobule, in part of the inferior parietal lobule, of the quadrate lobule and the gyrus fornicatus.

In a strictly practical field he had recently had a further confirma-

tion of these views. One of his patients at first had only ataxia and loss of the stereognostic sense, with impairment of all forms of cutaneous sensibility. Later there was added paresis and finally paralysis of the arm and leg and slight paralysis of the face, with some wasting, verbal amnesia, and temporary visual symptoms. In this case Dr. Keen had successfully removed a cystic tumor, which was mainly located in the superior parietal lobule, although it had grown somewhat forward and inward from this position. The diagnosis of a tumor in the superior parietal lobule had been made two months before the date of operation because of the ataxia and the sensory symptoms.

Dr. D. J. McCarthy asked Dr. Spiller whether the failure to trace the degeneration from the crossed pyramidal tracts into the internal capsule in a case of amyotrophic lateral sclerosis reported by Dercum and Spiller, was because the material was exhausted, or because there was no degeneration present. Also in regard to the presence of degeneration in the posterior longitudinal bundles. He also inquired whether Dr. Spiller considered the degeneration in the antero-lateral column a primary degeneration of motor fibers.

Dr. E. L. Mellus inquired in regard to the degeneration in the corpus callosum, and also whether it was possible to make out any grouping of motor focal points as suggested by Bevin Lewis' researches, and possibly by the fact that under electrical stimulation the motor points in man seem to be more separate than in the lower animals?

Dr. Spiller said that the posterior longitudinal bundles were slightly degenerated. The reason the degeneration was not traced above the pons in the case reported by Dr. Dercum and himself was because the degeneration ceased at this point and not because of lack of material. A similar condition has been observed in cases of parietic dementia. The degeneration in the corpus callosum was most intense in the middle portion of the part that is seen when the hemispheres are pressed aside. There was some degeneration in the knee of the corpus callosum, but degeneration in the splenium was not distinct.

With regard to the grouping of motor focal points within the motor cortex, as mentioned by Dr. Mellus, Dr. Spiller said he could not give any information. He had not been able to observe any difference in the degree of degeneration in sections taken from the same block.

With regard to the antero-lateral column, Dr. Spiller said that degeneration of this portion of the cord is not a very uncommon finding in amyotrophic lateral sclerosis. This degeneration may be in fibers connecting the cells of the anterior horns of different levels with one another.

21. EIN FALL VON DIFFUSER SARKOMATOSE DER GESAMTEN PIA MATER DES GEHIRNS UND RÜCKENMARKS (A Case of Diffuse Sarcomatosis of the Entire Pia of the Brain and Cord). Schröder (Monatsschrift für Psychiatrie und Neurologie, 6, 1899, p. 352).

The symptoms in this case were vomiting, choked disks, headache in the occipital and parietal regions, uncertainty of gait, exophthalmos, Romberg's sign, left facial paresis, deviation of the tongue to the right, diminution of the knee-jerk on one side and absence on the opposite, partial deafness, hallucinations of touch, etc. The cause of these symptoms was sarcomatosis of the entire pia of cord and brain, extending into the substance of the brain, cord, cerebral nerves and spinal roots. A brief review of the few cases of sarcomatosis of the central nervous system reported in the literature is given.

SPILLER.

## CHICAGO NEUROLOGICAL SOCIETY.

October 17, 1899.

The President, Dr. Richard Dewey, in the chair.

Neurological Observations in the Hawaiian Islands. Dr. D. R. Brower made remarks based upon a six weeks' visit in the Hawaiian Islands. He said in substance:

At the sea level the temperature is exceedingly equable, being in summer about 85, in winter about 80, with a difference between day and night of not more than 10 or 15 degrees; but owing to the great diversity in elevation and the constant presence of the trade winds, the islands as a whole present great variation of climate as regards both temperature and humidity. Along the coast the climate is depressing and predisposes to neurasthenia, especially among the women, so that they feel it necessary to periodically return to the continent for recuperation. The inference is that the islands would not be a good place to which to send neurasthenics, but the remarkable uniformity of temperature makes them a desirable place for the spinal sclerosis and for cases of interstitial nephritis. The mildness of the climate is also favorable to the treatment of insanity, as patients may be out of doors most of the time. In the Hospital for the Insane at Honolulu insanity among the natives was found to be of the milder types, mania being very infrequent, general paresis and paranoia unknown. Suicide is also practically unknown among the natives, the instances which have occurred being generally among the Chinese or Japanese.

Leprosy came to the islands from China in 1848. Despite the most vigorous efforts to stamp out the disease by segregation of its victims, it is still quite prevalent. At present there are two hundred lepers in the colony on the island of Moloki, where excellent provision is made for them: They occupy a tongue of land at one end of the island, separated from the remainder by a precipice three to five thousand feet high. Here they have a complete municipal organization, and are provided by the government with all necessities. Suspected cases are sent to a receiving station, where they are examined once a month by a board of health, unanimous action being necessary to consign a leper to the settlement. Doubtful cases are required to report to a health officer at stated intervals. The experts on the board have abandoned heredity as an etiological factor and regard the disease as contagious, accounting for its rapid spread and great prevalence among the natives by their habits of life. The principal article

of diet is eaten out of a common vessel with the fingers, which makes contagion almost inevitable. Among Anglo-Saxons the disease is infrequent. The proportion of the anesthetic form of the disease is not high, certainly less than 25 per cent. It usually begins with an erythematous eruption, the ulnar and peroneal nerves being the more frequent seats of the disease. At times the enlargement of the nerve is so considerable as to amount to two or three times the normal, the enlargement not being uniform, but nodular. In some cases the differentiation from Raynaud's disease is difficult. The period of incubation is long, five years being frequent, and much longer periods not unusual. The tubercular cases run a rapid course, while the anesthetic form is long-lived, there being at present in the settlement several cases which have lasted for more than twenty years. Hot baths and general tonic treatment seem to be the only remedies of any value; but some of the lepers who have gone to the mountainous districts of Japan have done so well as to be almost cured, although no authenticated instances of cure have been reported.

Dr. Henry M. Lyman thought that Doctor Brower's estimate of the temperature was a little too high. In a thermometrical record which he had kept for a year, the highest temperature was found to be 86, the lowest 60 or a little lower, and the average about 70 to 72. This was on the sea coast. That anemia is prevalent among the white women is very true, but among the men it is not more frequent than in other countries, and its prevalence among the women is, or at least was, due to the lack of exercise, opportunities for active out-door occupation for women having been few on the islands. Native women as well as the men are strong and vigorous. The prevalence of syphilis is something phenomenal, but the disease is very mild in its manifestations. When first introduced tuberculosis was exceedingly fatal and killed a very large proportion of the islanders, their mode of life being such as to conduce to the spread of the disease, the little huts in which they live being most unsanitary in every respect. When leprosy was introduced is unknown, as for many years it was mistaken for syphilis, everything of an ulcerative character having been referred to this disease. The methods thus far employed for the eradication of leprosy have been practically a failure because the natives have no horror of the disease, adopt no means to suppress contagion, and hide their leprosy friends and relatives from the authorities, sometimes for many years. The plan proposed by Hansen in Norway, that of the establishment of local hospitals throughout the country, is probably much to be preferred to a single large colony. The disease is not to any great extent communicable, and it is the prevalent belief on the islands that no Anglo-Saxon will become leprosy unless he adopts the manners and customs of the natives as regards promiscuity and uncleanness.

Doctor Brower, in answer to questions, said that there was only one physician in the leper settlement, and that this physician had never contracted the disease, but took extraordinary precautions to avoid contamination in any way. No data had been gathered in reference to central changes in leprosy such as occur in syringomyelia.

Dr. James G. Kiernan said that independently of the question of

leprosy Doctor Brower's interesting demographic paper deserved attention. The comparative infrequency of parietic dementia and locomotor ataxia, notwithstanding the unusual prevalence of syphilis, corresponds with what has been found in Egypt and British Guiana, where these two nervous affections are almost confined to the European population, although syphilis has been and is to-day very common among the natives. It would seem that in a people of intense commercial civilization, tendency to nervous break-down after specific infection prevails. With regard to another asserted factor in the causation of parietic dementia, it should be remembered that one of the most extravagant secret phallic societies has long existed, and still exists, in the Sandwich Islands, and it may be mentioned that Queen Lil's sister was notorious for voluptuous antics when a member of that society. Regarding the theory of Zambaco as to the relationship of leprosy of the attenuated type, Morvan's disease and syringomyelia, it may be recalled that in France leprosy long survived in a class of people who were separated from the mass of the population and called *cagots*, and it is precisely in those districts where these people were numerous that Morvan's disease has become frequent. The Arcadians were expelled from Canada largely for sanitary reasons, their descendants took leprosy with them to Louisiana, and the disease prevails there to-day, although it has not spread into other states. The disappearance of leprosy from European countries is still somewhat a mystery, and it is still an open question whether the English speaking people have not become practically immune to the disease.

Dr. Harold N. Moyer said that Hansen had asserted that leprosy is more common in America than we suppose, although the evidence adduced is far from convincing. Dr. Moyer had seen one case in this country in which the period of incubation must have been about ten years and which greatly resembled syringomyelia. The patient, a boy of eighteen, had the enlargement of the nerves, the characteristic anesthesia and erythematous plaques upon the shoulders and back, but these could only be seen when the patient was placed in a strong and direct light.

Dr. Brower, in answer to questions, said that near the receiving station for lepers a home for girls is conducted by the Sisters, where they have some thirty or forty girls born in the settlement, of leprosy parents, and although this home has been in operation for more than fifteen years, not a case of leprosy has developed among the inmates. Some physicians are inclined to think that the spread of leprosy was favored by the increased number of vaccinations; after one thorough vaccination of the entire population there was a very notable increase of leprosy.

#### A CASE OF BRAIN TUMOR.

Dr. Hugh T. Patrick exhibited the brain of a patient who during life had presented all the principal symptoms of general paresis, but had in addition well-marked weakness of the right arm, with repeated focal fits of this extremity. As he never had headache, vomiting, dizziness, tinnitus, or optic neuritis, tumor was excluded; and as he had contracted syphilis about nine years before, he was thought to have brain syphilis, probably syphilitic arteritis with thrombosis in the arm center, the cicatrix of which was the cause of the Jacksonian attacks. Finally, the patient had an apoplectic attack which



left him completely hemiplegic and aphasic, followed three weeks later by a second attack, from which he died after three days. At the autopsy a subcortical glioma was found, about the size of a hen's egg, occupying the centrum ovale beneath the middle of the posterior central convolution, which was atrophied and much distorted.

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22. COMMOTIONS DE L'ENCEPHALE ET DE LA MOELLE EPINIÈRE (Cerebral and Spinal Commotions). P. Manclaire (La Presse médicale, April 1, 1899, p. 153).

The patient, a locomotive engineer, was injured in a collision, and immediately became unconscious. Eight hours afterwards it was found that the muscles were flaccid; there was slight reaction to pain; the reflexes were preserved. He appeared plunged into a profound sleep, but raised the eyelids slightly when his name was called. During the night he vomited a number of times and there was incontinence of urine and feces. The pulse was hard and tense, and the pulse rate 62 per minute. The respirations were irregular, averaging about 28 per minute. Palpitation of the cranium was negative. From these symptoms the author believes that a condition of cerebral commotion was present, and excludes fracture of the skull or contusion of the brain. The second patient received a severe blow in the temporal frontal region, became unconscious, and subsequently passed into a state of partial coma. In this case also there was delirium, vomiting, incontinence of urine and feces. The pulse was slow, the respirations frequent. In the course of nine days, the patient was able to leave the hospital, but still at the end of two months complained of headache. The subsequent history of the first case was as follows: At the end of 24 hours the patient became partially conscious; subsequently entirely so. He then complained of headache, the pupillary reflexes reappeared, and improvement seemed established, when, 15 days after the accident, he had a renewed attack of vomiting, and on the 17th day a brief period of loss of consciousness. On the 21st day, however, he appeared to have totally recovered. The prognosis of these conditions is according to Manclaire rather favorable, but in patients with predispositions to these troubles there may be permanent intellectual disturbances, or a traumatic meningitis may occur. Two classes of theories exist regarding their nature: that they are some form of anatomical lesion, either minute hemorrhages, or degeneration of the cells and nerve fibers; or that there are no anatomical lesions, but merely disturbances of functional activity of the brain. The treatment should be expectant. Purgatives may be employed; but otherwise it is advisable to allow the patient to remain perfectly quiet. The author also reports two cases of spinal contusion; one occurring in a young man who was struck in the back of the neck, who lost consciousness for about ten minutes, and then re-awakened, complaining of intense pain in the back, which has persisted for six months without anatomical alterations or muscular atrophies. This could only be relieved by forcibly flexing the spinal column forward. In these cases prognosis is not quite so favorable, as there is the possibility of the development of diffuse myelitis. It is possible that these severe commotions of the spinal cord give rise to small hemorrhages; that is to say, in reality, contusions. SAILER.

## Book Reviews.

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THE NERVOUS SYSTEM AND ITS CONSTITUENT NEURONES. DESIGNED FOR THE USE OF PRACTITIONERS OF MEDICINE AND OF STUDENTS OF MEDICINE AND PSYCHOLOGY. By Lewellyn F. Barker, M.D., Associate Professor of Anatomy in the Johns Hopkins University, and Assistant Resident Pathologist to the Johns Hopkins' Hospital. With two colored plates and 676 illustrations in the text. New York: D. Appleton & Co., 1899.

ANATOMIE DU SYSTÈME NERVEUX DE L'HOMME. Par H. van Gehuchten, Professeur ordinaire à la Faculté de Médecine, Directeur de l'Institut Vésale. Troisième édition, premier volume, etc. Louvain: 1900.

The books of Barker and of van Gehuchten reached the reviewer's hands at about the same time. A comparison of the two was almost inevitable. Both of them were written by men now well known to the scientific world, men who have done considerable original work and who are most anxious to present the anatomy of the central nervous system in its newest light. Both authors have given great prominence to the modern neurone doctrine (no longer a theory, as van Gehuchten protests), with this difference, that the Belgian author has seen fit to describe the gross anatomy of the central nervous system according to the old and commonly received plan, whereas Dr. Barker has cut all bridges behind him and has started out upon the boldest attempt yet made to construct an anatomy of the central nervous system upon the basis of the "constituent neurones."\* While it may safely be said that the plan adopted by van Gehuchten is the easier one for those to follow whose knowledge of brain anatomy is based upon studies made years ago, Dr. Barker's book is truly remarkable for the enormous amount of special and detailed researches which he has embodied in its pages, and for the lucid manner in which he has discussed the conclusions reached by recent investigators. Both Barker and van Gehuchten are firm adherents of the neurone theory, as most of us may indeed be said to be to a more or less marked degree, though some feel that the neurone doctrine has not altogether passed beyond the stage of theory, and that there are certain anatomical and physiological laws which cannot be satisfactorily explained in accordance with it. Van Gehuchten accuses the opponents of the neurone doctrine, notably Apáthy, Bethe, Held and Nissl, of delighting to bring forth arguments against it, and of being somewhat over-zealous in speaking of the "mortal blow" which they have delivered at this theory. But the defenders of that doctrine (the neuronists!) seem to be no less ardent and hot-headed in their attempt to defend the theory at every point, and for the present it would appear that the burden of proof rests with them. It cannot be claimed that either van Gehuchten or Barker is convincing in his refutation of the results reached by Apáthy and Held. It may not be amiss to state in this connection that teachers of clinical neurology have not been overwhelmed by the neurone theory, as anatomists and physiologists appear to have been. The attempt to apply the theory to the elucidation of clinical types has

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\*Neurone is the correct term, according to Gildersleeve and Barker.

not been remarkably successful, except possibly with regard to the origin of *tabes dorsalis*. It is more than likely that the theory will have to be modified in some details before it can be raised to the dignity of a doctrine. Meanwhile it is to be regarded as a helpful working hypothesis.

In his remarkable monograph Dr. Barker has erected a veritable monument to this neurone theory. Should the theory fall at any time, the monument will prove a noteworthy tomb. In its outward appearance the American treatise strikes one by its entire lack of resemblance to previous works on anatomy. Only a few of the old-time illustrations are left, and were it not for the introduction of certain, now familiar, tables regarding the spinal functions, and of charts illustrating segmental representation in the cord, one would find very little to remind one of former text-books on anatomy. We cannot speak in terms of adequate praise of Dr. Barker's efforts to make the book thoroughly original. We believe, however, that if more attention had been given to the general gross anatomy of the central nervous system, the book would be more easily appreciated, notably by the large majority of practitioners for whom it appears to have been designed. With all due respect to the author and the practitioners, we ask where are the practitioners who have the time or inclination to read such books? Practitioners who consult a work of this kind would also wish not only to get the latest data regarding a single theory, but would desire, for instance, to get at the broad facts regarding the structure of the brain and spinal cord. Let us suppose that a physician were anxious to acquire the latest information regarding the sensory tract. He will find the information he wants in Section VI of Dr. Barker's work, and distributed therein in over 450 pages. He will find details which he cannot get from any other one book that has ever been published, but he will have considerable difficulty, unless he be almost as able an investigator as Dr. Barker himself, to form an adequate conception of the exact paths by which the sensory impulses travel from the skin or the special sense organs to the cortex. If a student of architecture wished to learn the fundamental principles of house-building he would probably not acquire them from a book devoted entirely to the system of planning, riveting and fastening the iron girders now in use in a modern building. The student of brain architecture, it seems to us, would experience very much the same difficulty in trying to form a conception of the "architectonics" of the central nervous system by a study of the "constituent neurones" and by chapters on the "grouping and chaining together of neurones." The facts, and the latest facts, are all there and are collated in masterful fashion; but one or two short sections, such as Edinger introduced in his book, on the general course of the chief cerebro-spinal tracts, would have added very materially to the value and usefulness of the work.

Dr. Barker's treatise is a splendid monograph on the histological structure of the brain and spinal cord. It will necessarily be a source of pride to all of us that such a work should have been published in this country, and if any error has been made in the construction, it is, in the tremendous importance that has been attached to a single doctrine to the exclusion of so many other fruitful theories and facts which have been gathered by the many able brain anatomists of the second half of the nineteenth century. The book is interestingly written, is profusely illustrated and should be in the hands of every working neurologist. The general practitioner who is able to enjoy it and to profit by it may well be considered to be above the average.

Of van Gehuchten's book we need merely say that the third edition

is an elaboration of the earlier ones, with an introduction of a very detailed account of the recent theories regarding the histological structure of the central nervous system. It is remarkable for its terseness and for the care with which contending theories and investigations have been considered. His opinion regarding the neurone theory he sums up by stating that the doctrine remains established, in spite of the assaults which have been made upon it from various sides. He thinks that one should be extremely careful and should view with a great deal of scepticism the opinion of the various authors concerning the existence of anastomoses. We must admit, he says, that the "réseau intracellulaire" described by Apáthy exists in reality. But to his mind the only fact which proves the correctness of the neurone doctrine is this, that when the axone is interrupted at any point of its course Wallerian degeneration sets in, affecting the peripheral end. This Wallerian degeneration and the subsequent atrophy in the cell body and in the central end of the axone stopped precisely at the point where the method of Golgi and Ehrlich have shown the limits of the neurone to be. If there were in reality the anastomoses which others maintain, why should this degeneration and atrophy not affect the neighboring nervous elements? The argument may be forcible enough, but it does not take us very much beyond the old theory that the cell body exerted a nutrient influence over the peripheral fibers directly connected with it. The natural inference from all this is that the neurone theory is not a finality; it leaves much to be explained regarding the structure and functions of the central nervous system.

B. S.

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#### MISCELLANY.

The next annual meeting of the American Medico-Psychological Association will be held in Richmond, Va., May 1, 2, 3 and 4, 1900. The secretary of the association is Dr. C. B. Burr, Flint, Mich.

The meeting of the International Congress of Medical Electrolgy and Radiology will be held in Paris, from the 27th of July to the 1st of August, 1900. All inquiries for further information should be addressed to Prof. E. Doumer, General Secretary, 57 Rue Nicolas-Leblanc, Lille.

The tenth annual meeting of the American Electro-Therapeutic Association will be held in New York city, on Tuesday, Wednesday and Thursday, Sept. 25, 26 and 27, 1900.

The officers of the Association are: Dr. Walter H. White, President; Dr. D. Percy Hickling, 1st Vice-Pres.; Dr. Charles O. Files, 2d Vice-Pres.; Dr. George E. Bill, Secretary; Dr. R. J. Nunn, Treasurer. Executive Council: Dr. Robert Newman, Dr. G. Benton Massey, Dr. William J. Morton, Dr. A. D. Rockwell, Dr. Charles R. Dickson, Dr. Frederic Schavoir.

THE  
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**Nervous and Mental Disease.**

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**Original Articles.**

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LANDRY'S PARALYSIS: REMARKS ON CLASSIFICATION.

BY E. W. TAYLOR, M.D., AND J. E. CLARK, M.D.

(FROM THE SEARS PATHOLOGICAL LABORATORY, HARVARD MEDICAL SCHOOL).

In spite of the fact that the complex of symptoms known as Landry's paralysis has been, during recent years, the subject of much critical work, there appears still to be opportunity for an analysis of the claims of the affection to be recognized as an independent disease. We offer the following case, therefore, typical in its clinical course, rather as a text for what is to follow than as a contribution of value to the extensive literature of the general subject:

The patient was a man of fairly strong physique, fifty-two years old. He was admitted to the Long Island Hospital, Boston Harbor, October 7, 1895. The antecedent history is unimportant as bearing on the present condition; he was not addicted to alcohol, and had had no venereal disease.

One month before entrance to the hospital the patient had had what he supposed was malaria, from which he recovered. Five days before entrance he was as well as usual. At about that time he stumbled and fell, without loss of consciousness. He was taken home, feeling weak. That night he had considerable pain in the back, and dorsal leg muscles. There was no headache. The chief complaint was a sense of weakness with occasional extreme pain, particularly in the legs. There was no bladder disturbance. A few days later he fell again in attempting to get off a car. He had, at this time, the same feeling of weakness he had before experienced, but was able

to walk about one hundred yards with the aid of a cane. The sense of weakness increased, and he was finally admitted to the hospital. On entrance he was found to be suffering from general weakness, considerable pain and a sensation of cold. The cranial nerves were at this time uninvolved. He remained essentially the same until October 9. At noon of that day a difficulty in articulation came on, then a gradually increasing disability to close the eyes; the following night he had some regurgitation of food and considerable frontal headache. Taste, smell and sight were unaffected; there was no diplopia. The general weakness was now so great that he was unable to stand unsupported. He complained of a numb sensation about the region of the stomach, and said that "his skin felt dead," below the level of the umbilicus. Bowels open.

*Physical Examination:* October 10. *Motion:* Excessive weakness; active and passive movements of the extremities free, but active movements executed with difficulty. Knee-jerks not obtainable; plantar reflexes absent; cremaster absent on the right and present on the left; abdominal and epigastric lacking. Arm reflexes normal. Considerable fibrillary tremor in the adductors of both sides. The patient is emaciated.

*Sensation:* Subjective complaint of cold, especially about the ankles. No pain on deep pressure over the nerve trunks; slight tenderness over the tendons of the dorsal thigh muscles. Sensation in the legs possibly slightly diminished to pin prick, but the point and head are correctly distinguished.

*Cranial Nerves:* Hearing, sight and smell unimpaired. Movements of the eyes free in all directions; the pupils react normally to light. Double facial paresis, more marked on the right side. Tongue protruded straight, but cannot be projected beyond the lips; swallowing imperfect; speech markedly affected, but no true aphasia. No mental disturbance whatever.

*Heart:* No murmurs; rapid; pulse 120.

*Urine:* Negative. *Temperature* normal.

Five days after entrance (October 12) a second examination was made in consultation with Dr. James J. Putnam. The general weakness had increased and slight sensory disturbances in the legs were noted, characterized by delay in perception and a failure to distinguish between hot and cold. The hands and face showed no abnormality. There was slight ataxia of the arms, but the hand-grasp was firm. The spleen was apparently not enlarged. (See autopsy note.) Electrical examination essentially negative.

October 17: Pulse 120-132; weak; tracheal râles. Speech more affected than at the last visit, but in the same manner.

Double facial paresis practically complete. Eyes glassy; cold sweating; urinary and fecal incontinence. Sensation to pin prick apparently still preserved; motor weakness extreme. Mental state clear; he is hopeful as to his condition. Died October 17, the same day, ten days after entrance to the hospital, and fifteen days after the first onset of symptoms.

*Autopsy:* 36 hours after death, October 19, under somewhat disadvantageous conditions. The body was considerably emaciated. The cord, brain, viscera, and following peripheral nerves were examined: the left pneumogastric, a portion of one sciatic, the left popliteal.

The cord microscopically showed no abnormal appearances; the membranes were normal. The skull and brain were normal, except for considerable opacity over the convexities, to which no pathological significance is to be attached. There was no increase in the amount of cerebro-spinal fluid.

Cultures were made at autopsy from the popliteal nerves and from the fluid about the cauda equina. Subsequent study of these cultures and the attempt to demonstrate bacteria in the tissues were rendered futile by the presence of contaminating organisms, probably putrefactive.

The chest cavity showed no pleuritic adhesions; the lungs were normal, except for a small focus, probably tuberculosis, at the right apex. Liver, heart and kidneys were normal in gross appearance. The spleen was considerably enlarged, weighing 360 gms. Intestines and stomach normal. No general arteriosclerosis.

Portions of peripheral nerves, a bit of the gastrocnemius muscle, the brain and cord, and a specimen from the kidney, were placed in hardening solutions preparatory to microscopic examination.

#### *Microscopical Examination:*

*Peripheral Nerves:* Marchi preparations: The sections examined show no characteristic staining of the myeline. The contour of the nerve fibers may often be made out, but the varicosities and stained fat droplets as seen in pathological conditions are absent. The same nerves stained by the Weigert method show considerable irregularity of staining. In view of the fact, however, that Marchi sections are negative and that the outlines of the Weigert stained fibers are regular, together with the relatively long time after death at which the specimens were hardened, lead us to think that what abnormal appearances are present are due to artifact, and are in no way to be regarded as pathological. Ventral nerve roots near the periphery of the cord show no abnormality. Dorsal nerve roots normal.

*Spinal Cord:* Sections cut from a large number of levels and stained by the usual methods. Unfortunately for the completeness of this report the Nissl method was not used, owing to the fact that the period after death at which it was possible to make the autopsy we felt too great to permit of definite conclusions being drawn from changes in the constituents of the cell body. The white matter of the cord as a whole shows no change whatever, so far as the nerve elements are concerned. The gray matter appears normal, except for a considerable degree of hyperpigmentation of the cells of the ventral horns, best brought out by staining with acid-fuchsin in aqueous solution. Many of the cells are almost completely filled with pigment, leading to an entire obliteration or marked excentric position of the nucleus and to a loss of cell structure. For example, in one group of twenty-five cells in the cervical region, ten show no trace of normal nuclei. Other cells of the same group are entirely normal in appearance, with well preserved processes and no dislocation of nuclei toward the periphery. The contour of these cells is regular, and many of them are noticeable from the fact of the small amount of contained pigment, in contrast with their engorged neighbors. So far as one may judge from this method of staining, the nuclei and chromatic portions of these cells are normal. This is, however, a point upon which we evidently cannot be dogmatic.

*Blood Vessels:* There is no evidence of arteriosclerosis; neither the larger nor smaller vessels of the cord show any true thickening of their walls. The smaller arteries throughout the cord, both in gray and white matter, are in many cases surrounded by hyaline material staining purple with hematoxylin, and remaining unstained with fuchsin and Weigert hematoxylin. This hyaline material apparently does not represent a degeneration of the vessel walls, but rather an extraneous deposit. In some instances the appearances are strongly suggestive of thrombi plugging the small arteries; in others the hyaline mass is evidently outside the lumen. There is also a certain amount of this same material lying free in the tissue and about the periphery of the cord; such deposits may occasionally be seen in the immediate neighborhood of nerve cells, in other respects apparently normal. There is no evidence of inflammatory change either in gray or white matter. The pia is normal in all the sections studied, excepting in the mid-thoracic region. At this point there is a typical tuberculous meningitis, involving the dura very slightly, but the pia throughout its whole circumference. The pia is everywhere thickened and infiltrated, the blood vessels are



involved, and the nerve roots, both ventral and dorsal, are invaded to a greater or less degree by the new-formed tissue. Giant cells and typical areas of necrosis are apparent. The periphery of the cord is invaded to a slight degree in typical fashion, but not sufficiently to interfere with general cord functions. We are unable to say how far downward this focus of tuberculosis extended, owing to the imperfection of the serial. There is no evidence of tuberculosis in the cervical region, nor in the oblongata, pons, cerebellum or cortex cerebri, so far as we have been able to study them, from the specimens at present at our disposal.

*Oblongata and Pons:* Nothing abnormal found.

*Cerebellum:* No pathological change discoverable, beyond the appearance of very numerous, usually round bodies, without concentric markings, and staining similarly with hematoxylin to those described in the cord, but much more regular in outline. These bodies are located in greatest numbers in the immediate neighborhood of blood vessels.

*Cortex Cerebri:* Similar structures are numerous, resembling those described in the cerebellum more closely than the irregular masses found in the cord. The cortex itself presents nothing abnormal.

*Spleen:* Simple hyperemia.

*Kidney:* Considerable degree of granular degeneration.

The search for bacteria as possible cause was unavailing, owing to contamination.

A summary of the findings in this case gives as positive result the presence of numerous hyaline bodies (amyloid) in the central nervous system, a focus of tuberculous meningitis in the thoracic cord, and changes of a pigmentary character in numerous ventral horn cells, studied particularly in the cervical region. The investigation is negative, as regards the discovery of degeneration in peripheral nerves, or positive evidence of the presence of bacteria as an exciting cause. Further changes in nerve cells than those noted above may have existed, but lack confirmation, because the Nissl method was unavailable.

An analysis of the lesions found, in the light of our present knowledge of so-called Landry's paralysis, will be of interest.

*Hyaline Bodies:* In 1891, E. Klebs<sup>1</sup> described a case of

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<sup>1</sup> E. Klebs. "Ueber Landry'sche Paralyse," Deutsche med. Wchschr., Vol. 17, 1891, p. 81.

Landry's paralysis, in which the essential lesion found was a hyaline thrombosis involving only the branches of the ventral spinal artery to the ventral horns. The nerve cells he found surrounded by a net-like substance, containing cellular elements. This he regarded as a transudation following thrombosis. The situation of the thrombosis only in the region of the ventral horns, he would explain by the anatomical arrangement of the vessels of the cord. The patient had a tuberculous pericarditis, resulting from perforation into the pericardial sac, of a caseous bronchial gland. The etiology of the thrombi is left in doubt; no tubercle bacilli were found in the vessels.

This case, which, so far as we know, is the only one of the sort in the now extensive literature of Landry's paralysis, is of interest in connection with the markedly hyaline condition of the smaller vessels observed in our case. As stated in the description of the microscopic appearances, the hyaline was in our case not so sharply limited to the distribution of a single artery as in Klebs' cord, since very similar conditions were found also in the cerebellum. Although in many instances there is in our specimens a strong suggestion of thrombosis, it is evidently not so conspicuous as in Klebs', judging from his description. The presence of hyaline-like material in the region about vessels, and also about nerve cells, is possibly what Klebs speaks of as a transudation following thrombosis. In any case the hyaline deposit is of the nature of an infiltration rather than of a degeneration of vessel walls. The larger vessels are not involved. Of interest also is the fact that in both Klebs' case and ours there was a latent tuberculosis, certainly in our case not sufficient to cause death, or symptoms of moment, though possibly associated in some way as yet unknown with the extensive hyaline formation. Admitting, as Klebs seems inclined to do, that there is a definite connection between the hyaline and the symptoms of ascending paralysis, we are at a loss to see how, in our case, such a relationship is to be maintained, inasmuch as the hyaline was not sharply limited to the ventral horns, and in any event cannot be conceived as productive of the rapidly fatal symptoms observed. A more reasonable supposition is to regard the hyaline changes as part of

a general process of as yet unknown etiology, and therefore an effect rather than a cause. It should be mentioned that at least two other writers, Baumgarten and Immermann, allude to the existence of hyaline deposits in cases of Landry's paralysis.<sup>2</sup> A paper by Spiller<sup>3</sup> is of interest also in this connection. He found a great number of amyloid bodies in a man of forty, dying of a disease resembling Landry's paralysis, and suggests that possibly we have not attached sufficient significance to the part such bodies may play in pathological processes. Certain of the bodies found in our case are not unlike those pictured by Spiller in his figures 3 and 4.

*Tuberculous Meningitis:* We have described a localized tuberculous meningitis of the cord in the thoracic region, with no other discoverable lesion of the same character in the specimens now at our disposal. A lesion of the lung, presumably of tuberculous origin, was also mentioned. The association of a tuberculous meningitis with the symptoms of ascending paralysis has not before been described, and very probably is to be looked upon merely as a coincidence. A review of previously reported cases shows the occurrence of tuberculosis in several instances, but certainly not in a sufficient number to be regarded as standing in constant causative relation to the symptoms. Nevertheless, in the light of the negative researches regarding etiology, it is not to be forthwith denied that the infection with tuberculosis may not in a predisposed individual lead to the outbreak of Landry's paralysis, in the same sense that syphilis does to tabes dorsalis. We have, however, no positive evidence, nor analogy, to substantiate this view, and we could, at best, merely regard tuberculosis as one factor in a multiple etiology.

*Nerve Cells:* We have noted changes in numerous cells of the ventral horns, which are not to be explained as senile (the patient's age was fifty-two), nor as due to the long period which elapsed between death and the fixation of the material.

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<sup>2</sup> J. J. Thomas. "Two Cases of Acute Ascending Paralysis with Autopsy." *Am. Jour. Med. Sciences*, Aug. 1898, p. 133. This paper contains an excellent digest of the literature up to 1898.

<sup>3</sup> Spiller. "On Amyloid, Colloid, Hyaloid and Granular Bodies in the Central Nervous System." *N. Y. Med. Jour.*, Aug. 13, 1898.

The changes are therefore positive, and may stand in causal relation to the symptoms, though we are more and more of the opinion that a conservative attitude toward the significance of cellular changes, such as are shown, for example, by the Nissl method, is in the present state of our knowledge the safest one to adopt. Pigmentation is notoriously a dangerous index of cell changes, since pigment is a perfectly normal constituent of many cells in the central nervous system. We are, however, safe in attributing to it a pathological significance, when it so predominates as to completely fill the cell body, particularly if associated with this hyper-pigmentation there be also a marked dislocation of the nucleus. The further and more important question of how such pigmentary atrophy is associated with the peculiarly rapid and characteristic symptoms of ascending paralysis remains absolutely unanswered, and we have no desire whatever to force the relationship. The point to be noted merely is that such cell changes existed in a nervous system which was also the subject of ascending paralysis.

The peripheral nerves, as far as our examination went, remained intact. We must, therefore, conclude that the lesions found in our case, however interesting from a general point of view, afford us little help in the interpretation of the symptom-complex observed during life, a result to which many other examinations of this so-called disease have also led. It is clearly one thing to find lesions, and quite another to associate them causally with symptoms. This is an error into which we are likely to fall, as our enthusiasm for the mere findings of the microscope increases.

It is quite superfluous to rehearse again the literature of Landry's paralysis. That has recently been done with close critical analysis by Diller and Meyer,<sup>4</sup> Bailey and Ewing,<sup>5</sup> Mills and Spiller,<sup>6</sup> and Thomas.<sup>7</sup> No doubt much has been gained by this careful work, and by other papers which have since been published, but still the opinions differ fundamentally as to the exact or even approximate nature of the disease, or

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<sup>4</sup> Diller and Meyer, *Amer. Jour. Med. Sc.*, April, 1896.

<sup>5</sup> Bailey and Ewing, *New York Med. Jour.*, July 4, 11, 1896.

<sup>6</sup> Mills and Spiller, *JOUR. OF NERV. AND MENT. DIS.*, June, 1898.

<sup>7</sup> Thomas, *Am. Jour. Med. Sc.*, Aug., 1898.

whether, in fact, it is after all to be regarded as an independent disease process or not. When such a diversity of opinion prevails at the end of a long period of conscientious study, we may assume that the various writers are using terms inconsistently, and with different meanings, or that we have not yet arrived at a fundamental conception of the process which lies at the root of the clinical symptoms, and hence are giving superficial facts an importance which does not belong to them. In the study of Landry's paralysis we have apparently fallen into both these errors.

Landry in 1859 described a series of symptoms, chiefly motor in type and usually fatal, which were quickly received into the long list of diseases as an individual entity, without pathological or etiological basis. The inevitable occurred. Conditions which showed pathological changes, with clinical course similar to that described by Landry, were still called Landry's paralysis, doubt merely being thrown on the earlier negative findings. These cases, which varied considerably from Landry's original type, were included in the same category, and the whole discussion about peripheral neuritis arose. If we mean by Landry's paralysis what Landry described, it is evidently absurd to arbitrarily extend the meaning of the term on the clinical side to various conditions which he did not describe. If the word is to be used at all as descriptive of a supposed disease entity, it must be used in the sense Landry meant it to be used and in no other, otherwise it would be far more conducive to progress if it be immediately dropped. Even a superficial study of the literature shows that the various writers on the subject have been absolutely inconsistent in the use of the term, "Landry's paralysis." The tendency has been to make the symptoms fit the pathological findings as these have grown more conspicuous and numerous, with what complete confusion we know. In other words, if Landry described a certain disease, it is clear that we must find the pathological anatomy and etiology of that supposed disease, and not of some other, or else give up the word as an unnecessary encumbrance to our already over-burdened nomenclature.

The classification of the group of symptoms known as Landry's paralysis becomes, therefore, of paramount importance.

if our studies are to lead to any profitable end. If we find that the various symptoms comprising the group are sufficiently coherent to make highly probable the final unity of the entire process, we are justified in maintaining the name; otherwise not. The first question to put to ourselves is: Are any of the individual symptoms described by Landry fundamental? Motor paralysis of a flaccid type beginning in the lower extremities and without involvement of the sphincters must now be looked upon as in great measure a fortuitous circumstance. Cases in which the upper extremities have been first involved have been described with, in other respects a typical symptom-complex of so-called Landry's paralysis. Leyden has seen fit to distinguish two forms, which he calls bulbar and neuritic, to overcome this difficulty of the direction of spread of the process. Hence, according to this point of view, only some cases of Landry's ascending paralysis are ascending. Landry was then wrong in laying stress upon this ascending characteristic of his disease. Certainly the flaccidity of the muscles and the non-involvement of the sphincters are of no consequence in establishing the identity of a new disease process, since they occur frequently in a variety of involvements of the peripheral motor apparatus. To go a step further, it seems that Landry was very tentative regarding sensation. In a case he described, sensation was definitely affected, and we have rather assumed that the opposite was the fact, because in many cases it has not been an obtrusive symptom. The frequently reported unaltered condition of the muscles, as regards atrophy, and the maintenance of unchanged electrical reactions are no longer to be regarded as significant of anything peculiar in the disease process beyond the fact that it is often very acute. In more prolonged cases, changes have often enough been found to throw doubt upon their absolute non-existence as a future possibility even in those cases which quickly result in death. Finally, to say that a disease is usually fatal does absolutely nothing to clear up the difficulty. It is not always fatal, we are informed, and even if it were, that fact alone would not mark it as in any way peculiar.

Evidently, then, none of the supposed classical symptoms of Landry's paralysis have, in the light of our present knowledge,

anything characteristic about them. As a group they are equally vague, and have no fundamental unity as an individual process. As long ago as 1876 Westphal, apparently seeing how diverse the speculations regarding the symptom-complex were becoming, attempted to define exactly what should be regarded as Landry's paralysis. To that end he made the statement that Landry's paralysis was characterized by its progressive, ascending, and finally fatal course; by the lack of electrical changes in the paralyzed muscles, and by the negative findings at autopsy, statements which succeeding investigators at once began to modify as too narrow to include all the cases. With this reaction, Landry's paralysis, as a clinical entity, to our minds, ceased to exist. We have since been talking about various more or less coherent symptoms vaguely resembling Westphal's conception, but always modified to suit individual needs. The most interesting, and in some respects the most significant, reaction against the early ideas, has been the insistence that everything is explainable on the basis of a peripheral neuritis. Ross inaugurated this movement in 1889, and since then it has found various adherents, who have easily elaborated many arguments in favor of their proposition. Very recently Walton<sup>8</sup> and Krewer<sup>9</sup> have again argued the point. Walton modifies, on the basis of a careful analysis of reported cases, the usually accepted conception of the process, and defines it as a form of toxic peripheral neuritis. This conclusion he bases on the report of a case of his own and a study of other cases, with reference particularly to disturbances of sensation and electrical reaction. Worcester,<sup>10</sup> in a later publication, is inclined to agree with Walton. Krewer is absolutely confident of his position, that Landry's paralysis is nothing other than the second and third stages of a chronic multiple neuritis, which through continuity of structure has invaded the spinal cord. He further thinks that a new factor usually in the form of an infectious disease, must supervene on a pre-existing

<sup>8</sup> Walton, Bost. Med. and Surg. Jour., Dec. 26, 1895. It should be stated that in a recent conversation with one of us, Dr. Walton said that he had changed his opinion regarding neuritis and Landry's paralysis. His present standpoint is not known to us.

<sup>9</sup> Krewer, Ztschft. f. klin. Med., 22, 1897, p. 115.

<sup>10</sup> Worcester, JOUR. NERV. AND MENT. DIS., May, 1898.

polyneuritis to induce the symptoms of Landry's paralysis. Four of the writer's cases are described in this paper; one of the patients lived and the other three fatal cases strongly suggest alcohol as an important etiological factor. Marked changes of a degenerative character were found both in the peripheral nerves and the cord, hence according to the author, Landry's paralysis is peripheral neuritis.<sup>11</sup> That patients die of rapidly progressive neuritis is unquestioned, but why call such cases Landry's paralysis, and then assume that we have discovered its pathological anatomy?<sup>12</sup> This is evidently a begging of the entire question.

We quote these facts merely to illustrate our point that Landry's paralysis as a circumscribed group of symptoms has come to grief. The tendency has become quite irresistible to group under this convenient heading various clinical phenomena, between which no adequate bond of union has yet been found. Under these circumstances it might be expected that post-mortem study would throw light on the matter, and supply the desired facts which should make of Landry's paralysis a separate disease process.

This hope is by no means justified by a study of the more recent literature of the pathological changes. So far as we are able to discover, there is simply nothing characteristic about any of the lesions described by various writers. The more we look the more we find, undoubtedly, but no one lesion or series of lesions is explanatory. Mills and Spiller,<sup>13</sup> for example, after an exceedingly critical analysis of the whole question, and on the basis of a carefully studied case, reach

<sup>11</sup> We confess to a certain scepticism of Krewer's pathological findings in general from the following statement, p. 123: "Im Endoneurium finden wir eine Hyperplasie der Neurogliazellen, ohne jedoch dieselbe als eine Zelleninfiltration ansehen zu können."

<sup>12</sup> A patient, for example of alcoholic tendencies under the observation of one of us at the Long Island Hospital, Boston, was taken with a gradually progressive muscular weakness, especially marked in the legs, associated with certain sensory disorders. The condition was progressive; the patient became almost completely paralytic and death resulted two months after the onset of symptoms. Were one so disposed, it might be possible to lay stress on the progressive motor weakness ending in death, and call the disease Landry's paralysis, instead of a general affection of the nervous system resulting from alcohol. We suspect that Krewer's cases were of this type.

<sup>13</sup> Diller and Meyer, *loc. cit.*



the following general conclusions: that a type of disease, as described by Landry, exists; that transitional forms occur, making the diagnosis between Landry's paralysis, polyneuritis and myelitis difficult; that it is possible in some cases that no lesions exist; that Landry's paralysis may be due to myelitis alone; that polyneuritis may be present, usually accompanied by changes in ventral horn cells; that in some cases the entire peripheral motor neurone may be attacked at the same time by the poison of the disease. It appears from these statements that Mills and Spiller are inclined to be liberal in the interpretation of the pathological anatomy of what they still call Landry's paralysis. Inasmuch as Landry's paralysis may be due to each of these lesions, it is apparent that it is not due exclusively to any one lesion, and hence lacks coherence from the point of view of its pathological anatomy.

Bailey and Ewing<sup>14</sup> conclude from their work that the commonest seat of the lesion is within the cord or oblongata, though it may be present in the cortex or in the nerve roots. They go on to say: "When in the spinal cord the lesion is that of acute anterior poliomyelitis—namely, an acute exudative inflammation, following the distribution of the central branch of the anterior cerebral artery, with cellular infiltration of the circumvascular sheaths, degeneration of ganglion cells, loss of structural elements, and with or without degeneration of the anterior roots." These writers are doubtful of the peripheral lesion, and admit that the disease may run a fatal course without demonstrable histological changes in the nervous system. This is a perfectly candid statement that Landry's paralysis is poliomyelitis in the majority of cases, which widens our pathological anatomy, but unfortunately renders still more vague our terminology.<sup>15</sup>

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<sup>14</sup> Bailey and Ewing, *loc. cit.*

<sup>15</sup> The following case, to be reported in detail in a subsequent paper, is of interest in this connection: A man of 25, previously well, after several days of vague discomfort, lost the use of his legs, and subsequently of his arms to a certain degree; sensation was only very slightly disturbed; the paralysis was flaccid. After three months, during which he improved slightly, he died. Autopsy showed a marked congestion of both ventral horns throughout the whole length of the cord, which microscopic examination proved to be due to a destructive inflammation. Nissl sections showed many damaged and some intact

Krewer, already quoted, is confident that Landry's paralysis is a subacute chronic polyneuritis (whatever that may be), and an acute diffuse degenerative myelitis. He, of course, substantiates his view by autopsy and microscopic examination. This is quite opposed to Bailey and Ewing, and, in fact, to most other observers, for however often peripheral degenerations have been found, a true neuritis has been exceedingly rare.

Meyer<sup>16</sup> makes the following pertinent remarks: "Landry's paralysis is a symptom-complex without quite uniform post-mortem findings. There may be cases of very rapid course in which the naked eye, and even microscopical examinations with the help of older methods, does not reveal any lesion. With the improvement of the technique, these cases have become very rare, and there is reason enough to believe that the most recent technical proceedings would have demonstrated changes in most cases. Hereby one of the most important items that induced Landry to describe this symptom-group as a disease, *sui generis*, has fallen; the post-mortem findings can no longer be said to be negative, but they are at least not quite uniform." This is a very tentative statement of what is becoming more and more apparent as investigation progresses. We would simply go a step further and say that the lack of uniformity in the post-mortem findings is much more conspicuous than the similarities. Meyer, for example, in this same paper, calls attention to a degeneration of the pyramidal tracts as one of the lesions, a fact, in itself, of great interest, but quite out of accord with our preconceived ideas of the pathological anatomy of the disorder. On the other hand, in spite of perfected technique, lesions are, in certain cases, still undiscoverable. A case of this sort is described by Girardeau and Lévi,<sup>17</sup> who, in spite of a complete examina-

cells. The diagnosis during life had been Landry's paralysis. According to Bailey and Ewing it was, but to our minds a better service would be rendered by calling it what the lesion demonstrated it to be, an acute poliomyelitis. Certainly a pathological term is to be preferred to a purely descriptive one, and that of highly inexact meaning. (Compare this case with one of those reported by J. J. Thomas, *loc. cit.*)

<sup>16</sup> Mills and Spiller, *loc. cit.*

<sup>17</sup> Girardeau and Lévi, "Un cas de paralysie ascendante aigue sans lesion histologique des nerfs et de la moelle." *Rev. Neurologique*, Oct. 15, 1898, p. 669.

tion, using the Nissl method, found nothing abnormal. Thomas<sup>18</sup> is inclined to the view that the essential feature of the process is an affection of the motor (spinal) neurone of the first order, which, however, may vary within very wide limits, depending upon the intensity of the infection. On the one hand these cases are very similar to acute anterior poliomyelitis, and on the other show changes of the slightest character in the motor elements. In general, Thomas thinks that the evidence "seems to justify us in retaining this collection of symptoms as forming a separate disease, clinically at least." This is a conservative statement of the case, and is merely open to the criticism that knowledge, on the whole, is not advanced by postulating groups of symptoms as diseases, particularly when it appears possible to discover an anatomical basis for such symptoms. In this matter of Landry's paralysis we are bound to find some essential connection between lesion and symptom, or else renounce the disease even as a clinical entity. This attitude the rapidly accumulating investigations on the subject are forcing upon us. We are still in the dark as to any fundamental and necessary association between the pathological findings and the observed symptoms.

It would be easy to multiply examples from the literature of discrepancies in the post-mortem appearances of the nervous system, in persons dying of what we have been in the habit of calling Landry's paralysis. The instances already given, however, seem quite sufficient to demonstrate the fact that there is absolutely no unity in these findings, and that, therefore, a causal relationship between them and the generally accepted symptomatology cannot be maintained.

In the absence of evidence tending to establish the individuality of the affection, both on the clinical and pathological side, it may be thought possible that a study of etiology will throw light on the matter, and supply the necessary link between cause and effect. This is, however, most certainly not the case. The vaguest part of the whole picture lies in the discovery of a constant etiological factor. To say that the disorder is due to an infection of some sort, or follows infec-

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<sup>18</sup>Thomas, *loc. cit.*, pp. 158-161.

tious diseases, does nothing to differentiate it from all the other recognized infectious processes, which give rise to totally different symptoms. Nor is our knowledge advanced by saying that it is a toxemia, unless we can also say what sort of a toxemia it is. These sweeping statements do little more than restate the problem in somewhat more exact language, but are not in the least explanatory of the peculiar symptoms which are claimed to be characteristic of the disease. The search for pathogenic organisms has also failed to show any relationship whatever between the bacteria found and the histological changes. Organisms described, for example, by Baumgarten, Centanni, Eisenlohr, and Piccinino, as occurring in the nervous system of patients dying of Landry's paralysis, are interesting as facts, but have as yet absolutely no etiological significance, for the reasons that they are not commonly found, that they vary widely in characteristics when found, and that they cannot be brought into causal relationship with the lesions or symptoms, by the recognized bacteriological experiments. As a matter of fact, beyond the mere statement that Landry's paralysis is probably due to a somewhat selective toxemia, our knowledge of the etiology does not go. The identity of the disease is rather dissipated than established by such a statement, inasmuch as it is thereby simply brought into the category of a great number of processes, which are awaiting completer investigation before they can be accorded the dignity of definite diseases, in the sense that diphtheria, for example, is a definite disease.

So-called Landry's paralysis is a particularly good example of a large class of disorders of function, having a certain superficial uniformity of manifestation in which observers have continually inclined to lay undue stress on similarities, and to ignore dissimilarities, in the absence of any fundamental criterion of comparison. On the basis of an acute clinical, and of necessity highly superficial, pathological observation made many years ago by Landry, subsequent observers, led largely by a name, have been attempting to force into the narrow clinical conception outlined by Landry various symptoms, none of them fundamental and supported by no constant pathological findings, and still less by a definite

etiology. This attempt, to our minds, has signally failed at every point, as we have attempted to show; the so-called disease has, with each new investigation, lost something of its coherence, when looked at from a critical point of view, and awaits a new classification under a broader conception of disease, for which our advancing knowledge is gradually preparing us. Evidently this new classification must be on the basis of etiology, and this applies not only to Landry's paralysis, but also to many other affections of the nervous system. One matter that must particularly impress itself upon the student of the pathological anatomy of the nervous system is the impossibility of sharply defining the limitations of anatomical changes in various text-book diseases. It is safe to say that such changes are always broader than the symptoms, which they are supposed to explain, as shown, for example, in the two well-marked diseases on the clinical side of tabes and paralytic dementia. We are too apt to be satisfied when we find alterations of structure, which we think explain our superficially observed symptoms, and too little inclined to search for symptoms of which pathological findings should lead us to expect the existence. A still commoner error, and one largely due to our defective knowledge, is our neglect of etiology. To define the cause of diseased tissues and abnormal symptoms is the ultimate work of the scientific medical investigator, and until that is done our classifications must not be dogmatic. Tuberculosis and many of the other germ diseases have yielded to this demand, with what result for exact medicine we know. For obvious reasons our knowledge of cause in the broad sense has lagged behind in relation to the nervous system, and we find ourselves postulating all sorts of diseases from symptoms alone, quite regardless of the broader conceptions which it should be the function of our scientific training to develop. However necessary this may be as a starting point of investigation, it is desirable to remember that conclusions based on such observation should always be tentative and not final. Landry's paralysis is a flagrant example of the failure to recognize this fact. We have persistently clung to a name and demanded that the symptoms which it represented should conform to our preconceived idea, rather

than be merged in a larger idea of the fundamental process. If this were done and the term "Landry's paralysis" dropped as indicative of a special form of disease, no doubt our progress toward a reasonably scientific classification would be materially advanced. Bright's disease has gradually given place to "nephritis," a bad term still, but a great improvement; why should not "Landry's paralysis" give place to an inclusive term of definite significance, which shall be broad enough to embrace the whole contradictory array of symptoms and pathological findings?

From the foregoing discussion we feel justified in concluding: that inasmuch as there is no essential constancy in the clinical symptoms, nor in the pathological findings of so-called Landry's paralysis, and inasmuch as the etiology is wholly vague, it is probable that the affection does not represent in itself a process to which the term "disease" may properly be applied, and that, therefore, it is desirable to drop the term, as unnecessary and misleading.<sup>19</sup>

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LE TRAITEMENT DE QUELQUES TROUBLES TROPHIQUES DU PIED ET DE LA JAMBE PAR LA DÉNUDATION DE L'ARTÈRE FÉMORALE ET LA DISTENSION DES NERFS VASCULAIRES (Treatment of Trophic Lesions of the Foot and Leg by Exposure of the Femoral Artery and Evulsion of the Vasa Vasorum). Jaboulay (Lyon Médical, 31, 1899, p. 467).

The above named surgeon is nothing if not original—not to say bizarre—in his contributions to surgery of the nervous system. In the case of an alcoholic and syphilitic man of forty-five years, who presented several small sloughing spots on the right foot and a cyanotic area on the right leg, all of three weeks' duration, and who suffered from pain in the second toe and in the leg, he exposed the femoral artery in Scarpa's triangle and tore away two nerves which accompanied the artery for about 15 cm. The following day a blister 5 cm. in diameter appeared on the sole of the foot, involved the entire arch, and broke on the tenth day revealing an ulcerated surface which was five weeks in healing. In the meantime, the original small sloughs separated and the ulcers healed. At the ends of six weeks the patient was discharged, the sores healed, but with pain in the second toe. The author's explanation of the result is quite as fantastic and immature as was the operation.

PATRICK.

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<sup>19</sup> Since this manuscript left our hands, a paper on Landry's Paralysis has appeared by Knapp and Thomas, *JOUR. OF NERV. AND MENT. DIS.*, February, 1900, p. 74. It is, in general, a reiteration of the opinions before expressed by one of the authors (Thomas), and in our opinion in no way controverts the position we have taken in this paper.

A CASE OF UNILATERAL PROGRESSIVE ASCENDING  
PARALYSIS, PROBABLY REPRESENTING A NEW  
FORM OF DEGENERATIVE DISEASE.<sup>1</sup>

BY CHARLES K. MILLS, M.D.

PROFESSOR OF MENTAL DISEASES AND OF MEDICAL JURISPRUDENCE IN THE  
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PHILADELPHIA HOSPITAL, ETC.

The patient, a man, 52 years old, was referred to me for diagnosis by Dr. Hugh T. Patrick, by whom and several other neurologists, and also by several ophthalmologists of Chicago, he had been carefully examined. About two years previous to coming under observation the patient's wife and some of his friends noticed that he occasionally stubbed his right toes and scraped his right heel, and gave other evidences of slight weakness or awkwardness of the right lower extremity. The patient was quite sure that the weakness in his leg came on slowly, the paresis up to the present time increasing with almost imperceptible gradations. It was not until the involvement of the right lower extremity had been apparent eighteen months that he noticed any weakness of the right arm. This weakness of the arm then appeared and soon became more and more evident, it being accompanied by a tendency to carry the arm rested against the body and flexed at the elbow. The paresis in the upper extremity had slowly become worse, although it had not nearly reached the degree of impairment observable in the leg. The patient can still use the arm for most purposes, but it soon aches from use. He can write slowly but with some difficulty and much aching. Up to the time of my examination his attention had not been called to any affection of the muscles of the face, but investigation showed some paresis in both the upper and lower distribution of the facial nerve; the forehead corrugated distinctly less on the right side than on the left in looking upward; the right face drooped slightly, its folds and contour being somewhat smoothed out. The time when the face was first involved could not be fixed.

About six months after the weakness of the right leg was first noticed the patient experienced much pain in the right gluteal region and upper and outer aspect of the right thigh. Later he had considerable hyperesthesia over the right lumbar and lumbosacral region, and two weeks later herpes appeared in this region with lessening of the hyperesthesia, although the entire outer and anterior aspect of the thigh now

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<sup>1</sup>Read before the Philadelphia Neurological Society, December 18, 1899. For discussion on this paper see p. 222.

became hyperesthetic. Shortly after the paresis in the arm was first noted he also had some hyperesthesia of the upper and outer aspect of the arm, but no herpes. The hyperesthesia of the lumbar region of the right lower and upper extremities disappeared in a few weeks.

The examination showed distinct wasting of the right lower extremity, apparently uniform throughout the limb, that is, not localized in any muscular groups. The measurements were as follows: Right thigh, seven inches above the middle of the patella, seventeen and three-eighths inches; left thigh, eighteen and five-eighths inches; right leg, six inches below the middle of the patella, twelve and three-fourths inches; left leg, thirteen and three-eighths inches. The measurements, therefore, showed a difference of one and one-fourth inches for the thigh and of five-eighths of an inch for the leg. The various movements of the right leg were distinctly weaker than those of the left, but were nowhere absolutely abolished. Similarly all the movements of the right arm were distinctly impaired, but were nowhere absolutely lost. The dynamometer showed 180 for the right and 160 for the left. Faradic contractility was retained. The affected limbs were not spastic nor contracted. Careful examination showed retention of all forms of sensation. The tendon and muscle phenomena on the right side were all somewhat exaggerated. Knee-jerk was plus on the left side, but was considerably more exaggerated on the right. Patellar clonus was present on the right but not on the left, and the right side showed a slight ankle-clonus, which was absent on the left. The plantar reflex was normal on the left; but on the right, while the Babinski reflex was not present, the normal response was distinctly less marked than on the left. It might be described as between normal plantar flexion of the toes and the dorsal flexion of the Babinski reflex. Ocular movements and pupillary reflexes were normal and ophthalmoscopic examination showed no changes in the fundus. The patient had lost his hearing in the right ear as the result of a shell concussion during the Civil War, but otherwise the special senses were normal.

My examination corresponded with the results obtained by Dr. Patrick and others who had investigated the case in Chicago, and who regarded it as purely motor.

This case, simple in its characteristics and easily described, represents a rare and possibly a unique form of disease. Between seventeen and eighteen years ago I saw a case somewhat similar in its features, similar at least during the months



that the patient remained under my observation. This patient was a woman, 43 years old, who three years before coming under my observation, while carrying her last child, began to notice weakness in her left leg, and soon had a slightly shuffling and limping gait. She had had some sciatica during her pregnancy. The left arm became noticeably paretic a few months after the left leg, and both leg and arm were in much the condition of the patient here described at the time when the woman was under my care. She had been examined by the late Dr. E. C. Seguin, who, in a note to me, said, "The presence of so much increase in the reflexes on the affected side would lead me to cling to the idea that there was a central (cerebral) lesion causing changes in the crossed pyramidal fasciculus analogous to lateral sclerosis." Neither arm nor leg was contracted during the time that the patient was under my own observation, although the paresis of the affected limbs slowly increased. Sensibility was preserved. The patient complained, however, that she had at times had pains which seemed like neuralgia in the limbs, more severe in the leg, the pain being relieved by a very hot foot bath. She also complained of what she called nervous twitchings in both the leg and arm, but more marked in the leg. The special senses were not impaired. She had slight impairment of her control over the bladder; as she expressed it, her urine was hard to hold. This patient passed from my observation, but was alive three or four years since, and was then affected in the extremities of both sides, but just to what degree and in what manner I have not had the opportunity to learn. She had, however, become entirely unable to walk.

Several diagnoses are suggested by a study of this case, as (1) an unusual form of unilateral disseminated sclerosis; (2) unilateral amyotrophic sclerosis; (3) a progressive hemiplegia due to slowly increasing focal cerebral lesion involving the motor subcortex or the internal capsule; (4) a degenerative motor neuritis; and (5) a functional hemiparesis; but the case does not seem to fit in exactly with any one of these diagnoses. A hemiplegic form of disseminated sclerosis has been described. Nodules of sclerosis appearing at successive periods in lower and later in higher levels of the pyramidal system

might be suggested as an explanation. A case has recently been reported which bears upon this diagnosis; but does not correspond exactly with the case reported this evening.

This case was studied in the Salpêtrière service of Prof. J. Dejerine, and has recently been recorded by Drs. A. Thomas and E. Long.<sup>2</sup>

The patient was a man 47 years old, with a history of having acquired syphilis at the age of thirty-six. At the age of forty, that is, seven years before his death, he began to experience progressive enfeeblement of the right leg, which proceeded gradually to complete paralysis with diminution of sensibility on the same side. Later he had incontinence of urine and feces. After a short stay at the hospital he improved, the amelioration in his condition being maintained until the end of 1894, when the paralytic symptoms reappeared in the right leg with also additional features of numbness and paralysis in the right arm. When he again entered the hospital in 1895 he had almost complete paralysis in the right lower extremity, with contracture on extension, the hemiplegic gait, exaggeration of the knee-jerk, and ankle-clonus, on the paralyzed side, and a slight degree of muscular atrophy on the same side. The knee-jerk was also exaggerated on the left side. The right upper extremity was a little paretic; the reflexes at the wrist and elbow were exaggerated on both sides, but more on the right. Sensibility was greatly diminished in the leg and trunk of the right side, while the right upper extremity presented a slight hypoesthesia at the level of the hand and forearm; the arm proper, the shoulder, the neck and the head were free from anesthesia. Incontinence of urine and feces persisted. The special senses were intact. His condition remained stationary until 1896, during which year he died, following an attack of acute pleurisy.

This case presents several points of difference from the patient reported this evening. Diminution of sensibility was present in the right leg from the first, and this went on until it was extremely marked in both the lower extremity and the trunk; hypoesthesia was also present in the wrist and forearm

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<sup>2</sup> *Comptes rendus hebdomadaire des seances de la Société de Biologie*, Oct. 13, 1899, 2 ser., t. 1, No. 28, p. 768.

after the arm became paretic. In my case no anesthesia has as yet been detected. Incontinence of urine and feces present, in the case of Thomas and Long, there have not been symptoms. My patient also denies any history of syphilis.

In the case of Thomas and Long a careful autopsy and microscopical examination were made. Several plaques of sclerosis were found. One of these was at the level of the fifth cervical root and occupied the entire right side, except a slight band of the antero-lateral cord. The plaque or nodule had disappeared entirely at the third cervical root above, and below at the level of the sixth cervical root. Another small plaque was found at the level of the seventh cervical root. This began at the dorsal part of the right column of Burdach and invaded the entire right dorsal horn as far as the base of the ventral horn. In the superior thoracic region some diffuse sclerosis was revealed, invading on the right the crossed pyramidal, direct cerebellar and ventral tracts; on the left the dorsal horn and tract of Gowers; and on both sides the columns of Goll and of Burdach, especially on the left. The same conditions were present in the mid-thoracic region.

The histological appearances are given in detail. The plaques of cervical sclerosis were notable because of the few vascular alterations. It is questionable whether the primary alteration was of the true nervous elements or of neuroglial tissue. The vascular origin of the diffuse sclerosis in the thoracic region appeared more probable, and recalled some of the lesions of spinal syphilis.

The ordinary train of symptoms found in disseminated sclerosis were not here present; but the lesions as described fairly explain the summary of symptoms given by the recorders of the case. The main feature of the disseminated sclerosis was its monoplegic form and the fact that the diagnosis was not made clear by intention tremor.

Several cases of unilateral amyotrophic lateral sclerosis have been put on record. In the paper read by Dr. Spiller at the last meeting of this society he referred to the fact that seven cases of the kind had been collected. Some of the diagnostic features of amyotrophic sclerosis are, however, wanting in our patient, and especially the absence of localized atrophy,

spasticity and contractures. It is not impossible, however, that these may develop later, and I cannot but feel that the case may represent a somewhat unusual form of amyotrophic lateral sclerosis. Indeed, the most probable diagnosis would seem to be that of a slowly increasing degeneration of the pyramidal fasciculi or of the cerebral motor neuron system.

A slowly increasing focal cerebral lesion does not seem probable, as such a lesion would not be likely to select for its slowly destructive effects first the motor fasciculi for the leg and then those for the arm and face.

Special and general symptoms of focal lesions, such as tumor, hemorrhage, softening and abscess are not present.

A degenerative motor neuritis is, of course, possible, but it is unlikely that such a peripheral affection would first select the leg, then the arm and then the face of the same side. The exaggeration of the deep reflexes is also an argument against a peripheral nerve affection.

The persistence and progressive increase of the symptoms and the absence of hysterical stigmata make a functional hemiparesis improbable.

Although some one-sided atrophy was present in this case, its other features do not seem to be in accord with the cases described by Brissaud<sup>3</sup> in discussing the cephalic trophoneuroses, which he shows are sometimes of cerebral origin, although the true trophic centers are situated at lower levels.

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24. WEITERE MITTHEILUNGEN UEBER VORSTELLUNGSREFLEX DER PUPILLEN (Report on Pupillary Reflexes). J. Piltz (Neurol. Centralbl., 18, 1899, p. 496).

As a result of many experiments, Piltz arrives at the following results. There are pupillary movements associated with psychical conditions (cortex reflexes, attention reflexes) that consist in contraction of the pupil when the attention is fixed on a bright object lying to the side of the line of vision, and in dilatation of the pupil when the object is dark. Furthermore, the mere calling up of images results similarly; that is when the image embraces objects, the pupil contracts and vice versa with an image of dark objects.

JELLIFFE.

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<sup>3</sup> Brissaud, E., "Leçons sur les maladies nerveuses," 2 s., Par., 1899.

A CASE PRESENTING RIGHT-SIDED HEMIPLEGIA WITH  
HEMIANESTHESIA, RIGHT HOMONYMOUS HEMI-  
ANOPSIA, JARGON APHASIA, WERNICKE'S PUPIL-  
LARY REACTION SIGN AND NEURITIC PAIN IN THE  
ARM OF THE PARALYZED SIDE.

By F. X. DERCUM, M.D.,

Clinical Professor of Diseases of the Nervous System, Jefferson  
Medical College; Neurologist to the Philadelphia Hospital.

The following case deserves to be placed on record because of the association of an unusual number of interesting symptoms:

A. H.; female; white; married; aged 62; a native of Germany and by occupation a washerwoman, was admitted to the Philadelphia Hospital, September 25, 1897.

It was learned that she had lost the use of her right side two weeks before admission, and that previous to this time she had always been a strong, hard-working woman. The right arm was completely palsied and flaccid. There was also loss of power in the right leg, although a slight degree of motion was observed at times. She was a large, muscular woman with skin and mucous membranes of good color. It was at once noted that she could not understand what was said, and that she could not make herself in any way understood. The pupils were equal and small; the pulse was of good strength and regular. In addition to the palsy of the right arm, the lower half of the right side of the face was also slightly palsied. Both knee-jerks were plus, as were also both biceps-jerks. Her tongue was heavily coated and her teeth covered with sordes. She was able to swallow without difficulty. There was no heart lesion save an accentuated second sound.

September 29—She was examined, and the following note was made: A typical right-sided hemiplegia was present. On attempting to move the arm a slight resistance to full extension was met with at the elbow. The paralysis of the arm was complete. The fingers were in a position of semiflexion. The paralysis of the right leg was also complete; the leg was flaccid, no resistance whatever to movements being met with. There was a slight drooping of the right angle of the mouth, but facial palsy was not pronounced. The tongue was protruded in the median line. Elbow-jerk and biceps-jerk were present and equal in both arms; not accentuated. A feeble knee-jerk was elicited in the right leg; in the left leg the knee-jerk was normal. On testing the patient for sensory loss no response was made to vigorous pin-pricks

over the right leg, right arm, right side of face and right side of trunk. The sensory loss was everywhere limited by the median line of the body. By carefully testing the patient, a right sided homonymous hemianopsia was also readily demonstrated. The pupils were equal, reacted to light and were rather small. The movements of the eyeballs, as far as they could be determined, were normal. A complete palsy of both sphincters was present. The patient was markedly aphasic, her vocabulary being almost entirely limited to yes and no. When asked her name, she repeated indistinctly her family name, Harenberg. She was unable to recall her first name, Augusta, but when Augusta was pronounced she at once recognized it. Tested with regard to taste she betrayed no sense of discomfort or look of intelligent recognition when tincture of nux vomica or other bitter medicines were applied to the right side of the tongue. When they were applied to the left side of the tongue she at once showed by her manner—by spitting and shaking her head—that she tasted the bitter substances. Similarly she gave no sign of recognition of any sensory impression when ammonia, valerian or other substances were held to the right nostril, though she appeared to recognize the presence of these bodies when they were held to the left nostril. Similarly she paid no attention when a loud ticking watch was held to the right ear, but at once opened her eyes, nodded her head and looked pleased when the watch was placed to her left ear.

For some time following this examination the patient's symptoms appeared to become more pronounced. The paralysis seemed to deepen and it also became more difficult to communicate with her. Examinations also seemed to fatigue her more readily than formerly. About one month after admission she seemed at times to recognize a decided tactile impression upon the right side, but sensory loss was still marked. She also manifested some return of power in the right leg. It was noticed also in testing the sensation of the right arm that pressure or handling of the arm gave rise to pain.

About October 28 patient developed great pain in the arm, and the nerve trunks in the right arm were everywhere excessively painful to pressure; especially was this true of the brachial plexus, and it was necessary to put the arm upon a splint. For a time the pain increased in severity and during the height of the attack the right hand became livid and edematous. In the course of a month the pain gradually subsided.

The patient having somewhat improved, she was gotten

out of bed and a systematic attempt was made to study her speech defect. It was found that she could understand very little what was said to her. In these tests her own language, German, was employed. She was able to recognize her own name. When asked to protrude her tongue or close her eyes she would comply after some hesitation; when asked to hold out her left hand she would at times comply and at times fail. By constant repetition a slight degree of re-education gradually took place, for instance she understood what was meant when she was told to close her hand and make a fist. She could not, however, distinguish between such words as "finger" and "thumb;" and again while she recognized the word "nose," she did not recognize such equally simple words as "chin" or "ear." She recognized the words "Germany" and "France" and when told she was French, shook her head in the negative. When asked whether she was from Germany, nodded her head and looked pleased. She frequently repeated the last word of the question that was asked her and frequently attempted a prolonged reply, the reply consisting of a series of *unintelligible but well articulated syllables*. Indeed this was the most frequent result on attempting to communicate with her. She evidently did not understand what was said to her; for instance, when asked where she was born, she pointed to her right arm and evidently attempted to enter into an explanation regarding her symptoms, stringing together a long list of unintelligible syllables. When asked to read her own name, the latter being written plainly in large German script, she failed to recognize it, but pointed with her finger at the paper and said, "Letters, letters." When told that the letters which spell Augusta stand for France, she nodded her head in the affirmative. Indeed she might be told that the letters meant anything and she seemed always to accept the statement. When asked to write her name, she held the pencil in her left hand and made an illegible and meaningless scrawl. Again when sentences likewise written in plain German script were held before her, she was utterly unable to interpret them. For instance she was asked in writing, "Are you from France?" She simply stared at the paper and manifested no response. If, however, she was asked in spoken words, "Are you from France?" she shook her head in the negative. She appeared to recognize that the paper contained writing, but that was all.

Her condition has undergone no appreciable change of late. It is the same as that noted at previous examinations. The hemianesthesia upon the paralyzed side, while less pronounced than in the beginning, is still readily demonstrated. It has changed very slightly within the past year. The loss of power

in the right arm and right leg is still very pronounced, the knee-jerks are both exaggerated and there is marked rigidity of the right leg. Paralysis of the sphincters persists. The apasia has changed very slightly, if at all. There is no optic neuritis.

Recently she was examined by Dr. Charles A. Oliver, who confirmed the existence of a right-sided hemianopsia and also demonstrated the presence of Wernicke's hemipupillary inaction sign.

The presence of the latter symptom is of great interest. In keeping with the profound hemiplegia (from which there has been no recovery) and with the persistent hemianesthesia, hemianopsia and sensory aphasia, this symptom indicates a most extensive lesion—one involving the entire posterior limb of the internal capsule, adjacent structures, such as the thalamus and radiations of Gratiolet and probably a large portion of the substance of the left hemisphere as far as the cortex. The lesion, judging from the history of the case, was doubtless vascular, though whether hemorrhage or embolism, it is difficult to say.

Regarding the neuritis which developed in the right arm no inference can be drawn. It is a rare sequel of hemiplegia, but has been seen by the writer several times. No adequate explanation has ever been advanced as to its origin.

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25. EPILEPSIE RETROPULSIVE (Retropulsive Epilepsy). M. Lannois (Lyon médical, No. 30, 1899).

The author calls attention to a rare manifestation of epilepsy, *i. e.*, the tendency to walk backwards before or during an epileptic attack.

The first case was that of a gardener, who had a large number of epileptic attacks, in many of which he retained consciousness, and in all of which he walked backwards ten or twelve steps. There were no grand convulsive seizures. The second case was in a tuberculous patient, who, before the convulsion occurred, would walk backwards three steps and then fall, either forwards or backwards. The autopsy revealed no cerebral lesion.

The third and most interesting case is that of a woman aet. 46. She had convulsions in infancy, followed by a right external strabismus. The convulsions and various automatic movements began at 17. She has daily convulsions. Very often she has peculiar attacks in which she becomes pale, rises from a sitting posture and walks backwards until she comes to an obstacle, when she begins to take her clothes off. If interrupted in this procedure she becomes angry and utters piercing cries. There are no convulsive movements of the limbs, but some twitchings of the facial, jaw and eye muscles, and frothing at the mouth. The attack lasts usually from five to fifteen minutes. She remembers nothing of the attack.

McCARTHY.



A CASE OF ACUTE POLIOMYELITIS ANTERIOR IN A  
YOUTH SEVENTEEN YEARS OF AGE.\*

BY WHARTON SINKLER, M.D.

Acute poliomyelitis attacks individuals most frequently between the second and third years, and after the tenth year it is comparatively rare, although it may occur at any period of life, and has been observed as late as sixty-two years. Of 609 cases collected by Starr from tables compiled by Seeligmuller, Galbraith, Gowers, and myself, 118 occurred during the first year of life, 214 in the second year, and 140 in the third. In this table cases were recorded with diminishing frequency up to the tenth year, at which age there were only six cases.

The etiology of the disease has not become any more clear during the past few years than it was when the affection was described by Duchenne as "Acute Atrophic Paralysis." There is now a general conviction among neurologists that poliomyelitis anterior is due to some infectious process, but what the nature of the infection is no one has demonstrated. The fact that several epidemics have occurred in certain localities is undoubted proof of the infectious character of the disease, although it must be said that the general symptoms and course of the affection in the epidemics are slightly different from those in isolated cases. An infectious process would explain, in a certain way, the reason for the fact which I pointed out many years ago that a large majority of cases of poliomyelitis occurred during the hot months of the year, and all of the epidemics of poliomyelitis which have been recorded have occurred during the summer: For example, the epidemic recorded by Caverly, of Rutland, Vt., occurred between the 20th of July and the 20th of September, 1895, and 144 cases, both in children and adults, were reported by him. Nearly all writers on the subject have regarded exposure to cold or sudden checking of perspiration as one of the causes; and in the patient before you this was apparently the occasion of the disease.

The most recent investigations into the pathology of the

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\*Read before the Philadelphia Neurological Society, Dec. 18, 1899.  
For discussion on this paper see p. 220.

disease show conclusively that it is primarily a vascular affection, and that the changes in the large multipolar cells of the anterior horns are secondary to vascular disturbance. In the early stage of the disease, when the opportunity for post-mortem examination has occurred, the blood vessels are found distended, and some of the capillaries are ruptured, allowing extravasations of blood cells; then follows a localized myelitis in the gray matter of the cord. The regressive character of the paralysis can surely only be explained on the theory of vascular changes, which are only temporary. It is interesting to observe that this view of the pathology of poliomyelitis, which was held many years ago, and was subsequently abandoned, has been re-established. A more comprehensive view as to the nature of the disease has undoubtedly been helpful in regard to the treatment of these cases. Remedies like the salicylates, which are indicated in the infective processes, have proved of great advantage in my hands in recent cases of poliomyelitis. In a child of three years of age, seen by me during the past summer, with paralysis of the left leg, the result of poliomyelitis, almost complete recovery has taken place. The patient was kept at rest for several weeks, and salicylate of sodium and ergot were administered. The case which I will present for your inspection is as follows:

J. N., aged 17 years, of New Egypt, N. J., was admitted to the Orthopedic Hospital and Infirmary for Nervous Diseases November 21, 1899. He was a farmer by occupation. His father and mother are living and well, as are five brothers and six sisters. There are no hereditary tendencies to paralysis, and there is no personal history of syphilis. He has always been well and vigorous. He had appendicitis four years ago. He was treated medicinally and was in bed for several days, but recovered completely. He had a fistula in ano, which made its appearance about a year ago, but it was small and never very troublesome.

On or about August 15, 1899, he went swimming in a lake, and remained in the water for at least an hour and a half. The day was cool, and soon after coming out of the water he had a chill, which lasted for a few minutes. He worked the next day, feeling as well as usual, but in three or four days he began to have headache, vertiginous sensations, and felt bad generally. For three or four days he had severe

pain in the back and in the hips, and was confined to bed. He then found that there was some loss of power in both legs, from the hips down. This was first observed about one week after the day on which he went in swimming. Muscular weakness was first noticed on attempting to go up stairs, when he found that he could not walk up the steps. The next morning he could stand only with assistance, as the legs would give way from the hips down if he was not supported. The pains had become much less by this time, and there was no trouble with the bladder or rectum. After a few days he was taken out of bed and allowed to go out on the porch. As a result he apparently caught cold, and there was return of the pains in the back and limbs. There was then retention of urine for two days, but he was catheterized only once. There then was found to be paralysis of both legs. The loss of power was complete in the left leg, but in the right leg he had some power of moving the toes; with this exception the right leg was powerless. There was no weakness whatever in the arms. In two or three days he began to recover from the pains, and in a week's time there was return of power in the right leg to a considerable extent.

He was admitted to the Pennsylvania Hospital on September 7, where he remained until November 14, and while there the fistula was operated upon and healed promptly. On admission to the Infirmary for Nervous Diseases, November 21, the patient was able to walk on crutches, supporting his weight on the right leg. He was able to move the toes of the left foot, but could scarcely stir the foot. He could partially flex the leg at the knee, but could not lift the leg so as to raise the heel from the bed. He had power of moving all the muscles of the right leg, although without vigor. The knee-jerks on the right side were present, but on the left side they could not be elicited. There were no changes in sensation, and there was no pronounced atrophy in the muscles of the legs. There was no loss of control of the bladder, and the bowels moved regularly. Examination of the urine gave negative results. Electrical examination was made by Dr. H. P. Boyer, who made the following report:

| Left.  |                  | Right.            |                         |
|--------|------------------|-------------------|-------------------------|
| 12 ma. | K.C.C.; 7 A.C.C. | Rectus femoris.   | 8 ma. A.C.C.; 7 K.C.C.  |
| 12 ma. | " "              | Flexors of thigh. | 12 ma. K.C.C.; 7 A.C.C. |
| 12 ma. | " "              | Adductors.        | 6 ma. K.C.C.; 7 A.C.C.  |
| 7 ma.  | K.C.C.=A.C.C.    | Anterior tibials. | 6 ma. A.C.C.; 7 K.C.C.  |
| 14 ma. | K.C.C.; 7 A.C.C. | Calf group        | 10 ma. K.C.C.; 7 A.C.C. |
| 12 ma. | K.C.C.; 7 A.C.C. | Peroneus          | 9 ma. K.C.C.; 7 A.C.C.  |

"The faradic response is lessened in all of the muscles of the legs. The left anterior tibial responds but very slightly

to the strongest faradic current. The right anterior tibial responds nearly as well as the other muscles of the legs to faradism. There is R. D. in right rectus femoris and in both anterior tibials."

The patient was kept in bed and massage and galvanism were thoroughly and carefully applied to the legs, and there has been a steady improvement in his condition. He can now move the right leg vigorously, and has power of flexion and extension of the left leg. He can also raise the whole leg without flexing it at the knee, and can flex and extend the toes, but has little more movement of the foot than he had originally. There is distinct wasting of the anterior tibial group of muscles in the left leg and of the quadriceps extensor, but the remaining muscles are so well preserved that there is no difference in the circumference of the two legs.

There are three points of interest in this case: First, the unusual age at which poliomyelitis occurred. Secondly, the fact that, although there was reaction of degeneration in the anterior tibial muscles of the left leg there was still good response to the faradic current, and that, although all of the muscles of the right leg have regained almost the normal amount of strength, there still remains reaction of degeneration in the anterior tibial group. The third point which is notable is, that there is exaggerated knee-jerk in the right leg, whereas two months ago this was entirely absent. I have seen one other case in which after recovery from poliomyelitis there was exaggerated knee-jerk.

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26. NEWSPAPER SCIENCE. Professor James H. Hyslop (*Psychological Review*, Vol. 7. No. 1, Jan., 1900, p. 64).

This is a note which, in justice to a competent scientific investigator, should be given all convenient publicity. Professor Hyslop disclaims any expectation of "scientifically demonstrating the immortality of the soul" as the newspapers said. Scientific people, as well as the multitude, read the papers, and it is these especially he would ask to suspend their judgment on this interesting matter—what he will have to say "should not be estimated from the standpoint of fakirs and newspapers." "Spiritism must either be accepted or killed—sneering is no longer effective or scientific." "Whether I am right or not I shall leave to others to decide." All that Professor Hyslop seems to ask is a *hearing* at which sensational newspaper ingenuity shall not wholly have the floor.

G. V. N. DEARBORN.

## TRANSIENT REAL BLINDNESS IN HYSTERIA.\*

BY GEORGE C. HARLAN, M.D., OF PHILADELPHIA.

Mrs. K. B., a delicate and nervous woman, 23 years of age, came to the Wills Hospital complaining of loss of vision in the left eye of three days' duration. She said that a month before there had been double vision in each eye, which lasted three weeks, and was succeeded a few days later by almost complete blindness in the left.

She had a good deal of frontal headache and intermittent pain in both eyes. The right eye had full vision and was normal in all respects. In the left vision was reduced to the ability to count fingers at four inches. A mydriatic had unfortunately been used before I saw her and prevented investigation of the pupil, but the ophthalmoscopic appearances were quite normal, excepting, perhaps, a slight retinal hyperemia. Various prism and confusion tests failed to elicit any higher degree of vision than she at first admitted. There was decided inversion of the blue and red fields, as is shown by this chart, which was carefully taken by Dr. Zuill, and also concentric contraction.

At her next visit, three days later, there was absolute monocular blindness. No diplopia was produced by flame and prism. The application of the modified Grafe prism test, suggested but not correctly understood by v. Welz, viz., watching the behavior of the eyes behind a prism, seemed conclusive. The patient was directed to look at a flame, and a prism, base toward the temple, was placed before each eye alternately. When it was held before the blind eye there was no movement, whether the other was closed or open; but when it was placed before the right eye there was very evident deviation of both eyes to the left and recovery after removal of the prism.

I believe that this test is absolute, as the turning of the macula to the fixing point is practically a reflex act over which the subject has no control. Priestly Smith has suggested that it is applicable even when binocular blindness is simulated, and Dr. Jackson has shown that in binocular vision only the eye before which the prism is held deviates, as the other is fixed by the flame, while in monocular blindness both eyes deviate, as in this case, when the prism is held before the seeing one. Of course, in case of simulated monocular blindness, we determine in this way only the fact that the alleged blind

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\*Read before the Section on Ophthalmology of the College of Physicians.

eye has light perception; how much it can see must be discovered by other means.

The patient did not return to the hospital for ten days, when she joyfully announced that the sight was rapidly returning to the blind eye, and easily read with it No. LXX. of Snellen's types at twenty feet. We had no further opportunity to study the case, as the patient disappeared. She probably recovered entirely, or turned up with a new set of symptoms at some other hospital.

It will hardly be questioned that this was a typically hysterical case; and I am equally satisfied that the eye was really and absolutely blind. It has interested me particularly because in a very considerable experience with cases of this kind I have met with but one other in which I have been unable to demonstrate that the blindness was not real, and have usually succeeded in making the patient read with the alleged blind eye.

In a paper read before the Philadelphia Neurological Society some years ago, I suggested that, from a clinical point of view, it seemed possible to divide these mysterious cases of so-called hysterical blindness into two classes. In some there seems to be a more or less deliberate deception, the result of a morbid craving for sympathy, or for personal importance, or of a motiveless freak of a disordered mind. The blindness may be considered a symptom of a kind of insanity—"ganglionic insanity," if you choose—as hysteria has been called. These patients are like the fasting girls who develop a superhuman ingenuity in the effort to make it appear that they live without eating.

Others seem to be quite honest and to be themselves deceived as thoroughly as they would deceive others. It is possible that though they see well enough unconsciously, they may not be capable of conscious vision. Our daily experience, when we use the microscope or ophthalmoscope with both eyes open, shows that, under certain conditions, the brain may take no notice of what the eye is seeing; or, rather, of the images formed upon a healthy retina with its nerve connections sound. Perception is in abeyance. Something similar may happen in the case of hysterical blindness. Just how or why, nobody knows. Call it "psychic" blindness, if you

will; though that term is scarcely more explanatory than is hysterical.

I am half tempted now to add a third class, in which blindness is real and absolute, but transient, and without recognizable cause.

27. "PSYCHOLOGICAL ATOMISM. Hugo Münsterberg (Psychological Review, Vol. 7, No. 1, Jan., 1900, p. 1-17).

This ingenious article will probably advance the science ultimately, in one way or another, more than will any other of its speculative achievements in months, for it is vital with the psychologic spirit of the hour—parallelistic, analytic, and scientific.

Having pointed out the success of analysis in the affective modes of consciousness as well as in the cognitional, the writer draws attention to the fact that in trying to determine the aspect of psychological correlation it is no longer the purely "material" or physical stimulus with which (as in Fechner's abandoned "law") the sensation is considered to be correlated, but that it is rather with that still indefinite commotion which is concomitant in the neural structure. "We have been too long satisfied with the hypothesis that the elementary stimulus of the outer world excites merely the physiological unit in the brain which is represented by the isolated ganglion cell. \* \* \* \* We are becoming more and more accustomed to the view that the most elementary stimulus brings about a nervous excitement which grows from the periphery to the center like an avalanche and stimulates in the brain a whole area which may overlap the area stimulated by another isolated excitement. \* \* \* \* As soon, therefore, as it is recognized that the brain-effect of the indivisible stimulus is an easily divisible physiological process, the sensation ceases to be the ultimate result of psychological analysis, since a still more elementary psychophysical unity can now be conceived as the last factor in the explanation."

"I am convinced," says the writer, "that the time has come when we need a psychological atomism just as much as an atomism of the physical universe." Professor Münsterberg's atoms then are elements which, while under the general conditions of consciousness, are different from our sensations, they are absolutely dissimilar to each other (for on such uniqueness alone can the empirical differences in the vividness of sensations depend); they can vary through all degrees of vividness; they are all co-ordinated, not being in special groups according to our different senses; and lastly, they by their manifoldness are the material out of which is built both the "so-called qualities" and the intensities of the sensations. All the differences in consciousness seem to Dr. Münsterberg explainable by the changes in vividness of his atoms.

"We must begin to see that the sensations are unsatisfactory as ultimate conceptions; and that they must be replaced by psychical atoms if psychology really desires to become worthy of its great older sisters, the natural sciences"—not, we presume, that the author deems psychology at present an unnatural science, or even that it is too largely metaphysical, if, possibly, too "mystical."

G. V. N. DEARBORN.

NEW YORK NEUROLOGICAL SOCIETY.

February 6, 1900.

The President, Dr. Frederick Peterson, M.D., in the chair.  
SUCCESSFUL TRANSPLANTATION OF TENDONS IN A  
CASE OF INFANTILE CEREBRAL PALSY.

Dr. B. Sachs presented a boy of fourteen years, who had had a typical cerebral hemiplegia, due to difficulties at the time of his birth. The boy had called at the clinic because of contracture of the arm. A transplantation of tendons had been done by Dr. W. R. Townsend, with a very gratifying result. These cases seemed to Dr. Sachs to hold out even a better prospect of recovery than the ordinary infantile spinal cases, because in spite of the contractures much more power is left in the various groups of muscles.

Dr. W. R. Townsend exhibited two photographs showing what had been the position of the hands and the limbs and the condition of extension and flexion of the fingers and wrists at the time the boy had first come under his observation. The method selected had been to transplant the extensor tendons on to the flexor tendons. The electrical reaction of the flexors was diminished. The anterior pair of the four muscles had reacted slightly. On December 21, under ether anesthesia, an incision had been made in the middle of the flexor surface of the arm over the wrist; then the flexor carpi radialis and flexor carpi ulnaris were divided above the wrist and held temporarily by silk sutures. The next step had been to make an incision on the dorsum of the wrist, and expose the extensor communis digitorum one inch and a half above the wrist. A dissection had then been made between the bones so as to connect with the tendons previously cut. The extensor communis digitorum was folded back, as it was too long, and the transplantation to the attachments of the flexor carpi ulnaris, radialis and palmaris longus was then made. All dressings had been removed at the end of six weeks, since which time the arm had been carried in a sling. The arm was now held in a position midway between flexion and extension, and there was a fair grasp of the hand, though there had been none before the operation. Before the transplantation the hand had been helpless and useless. If proper exercises were used the boy would be almost sure to have a useful hand, and would be able at least to use a pen.

Dr. Leonard Weber presented a case for diagnosis as to the mental condition—a man thirty-five years of age, a clerk



by occupation. The family history showed a marked tendency to mental disorder. In the spring of 1895 he had begun to be troubled with slight vertigo, drowsiness, headache and occasional fainting spells. He had then become somewhat erratic in his habits, and on August 10 had developed an attack of acute mania. In November Dr. Weber had first seen him while the patient was in a private asylum. In December he had been transferred to the Bloomingdale Asylum, and in the following May had been discharged, the physicians there looking upon the case as one of acute mania that had recovered. Some months later the man had exhibited symptoms of malarial infection and had responded fairly well to quinine. He had then been put tentatively on mixed treatment, but with negative result. Early in December, 1896, he had had a slight serous pleurisy. Some time afterward Dr. Weber had located a sub-phrenic abscess, and, on incision, over one pint of pus had been evacuated. The man's recovery had been uneventful, and on January 1, 1897, he had resumed active work, and had continued at his occupation ever since that time. The patellar reflexes had diminished, and sexual power had been lost. The case was presented with the idea of determining the cause of the mania—whether it had resulted from an infection.

#### AMYOTROPHIC LATERAL SCLEROSIS.

Dr. E. D. Fisher reported the case of a woman, forty-nine years of age, who gave a history of mental disorder in her immediate family. The patient had presented no evidence of syphilitic infection. She had been very ill in each of her two pregnancies, and after the last confinement, which had occurred sixteen years ago, she had suffered from neuralgia of the leg. In December, 1898, she had noticed some difficulty in talking, and ten months later speech had entirely failed. About this time she had first noticed some difficulty in passing the bolus of food into the back part of the mouth. On December 20 she had come under his observation. At that time there had been inability to speak or swallow, and the jaw was rigid. The tongue was small and short, and rather rigid. Examination showed the same rigidity throughout the muscular system. The first diagnosis had been bulbar paralysis. The tone of the voice was now entirely nasal; the tongue was small, and the muscles of the neck and jaw so rigid that the mouth could only be opened slightly. There was an increase of the reflexes. Swallowing was difficult. The rigidity of the tongue and the increase of the reflexes were the special features of the case.

Dr. C. L. Dana suggested that the case was one of amyotrophic lateral sclerosis.

Dr. Joseph Collins concurred in this diagnosis, and said that he had reported an exactly similar case in 1892, the spastic condition of the neck and face having been the most pronounced and the initial symptom.

Dr. Fisher said that he would accept the diagnosis suggested by the last two speakers.

#### DIPLEGIA BRACHIALIS TRAUMATICA.

Dr. Pearce Bailey presented a man, fifty-four years of age, who had been knocked down and injured by a carriage last December. Two ribs had been fractured, and both upper extremities had become paralyzed. He had been treated for a short time at the Roosevelt Hospital. On coming under observation in January, 1900, examination showed the movements of the neck to be free and painless. The muscles about the shoulders were paralyzed and atrophied, and responded sluggishly to faradism. He complained chiefly of severe pains running down from the shoulders into the arms. There was no paralysis of the legs or of the bladder, and no anesthesia. The diagnosis was diplegia brachialis traumatica. Kocher had maintained that this condition was the result of an extra-dural hemorrhage, basing his opinion apparently on the existence of the darting pain down the arm. Dr. Bailey thought there might be compression of the nerve roots, or hemorrhage into the cord itself. He did not think an extra-medullary hemorrhage would cause such symptoms as existed in this case, and moreover such a lesion occurring as an isolated one is practically unknown.

Dr. Max G. Schlapp thought if the lesion were due to hemorrhage into the cord there should be more marked sensory disturbance.

Dr. Collins thought the chances were greatly in favor of this condition being one of hematomyelia. It did not seem to him at all probable that the rapid atrophy of the muscles could have resulted from an extra-medullary lesion; it was rather a true atrophic condition dependent upon the destruction of some of the motor cells at their origin.

Dr. E. D. Fisher was of the opinion that there was not a destructive lesion in the anterior horn, but slight capillary hemorrhages.

Dr. Schlapp thought the hemorrhages must have been on both sides in the anterior horns, and if they had been so large as to cause such atrophy they must have affected also the anterior commissure and the gray matter of the anterior horns.

Dr. Mary Putnam Jacobi did not see why there should be any more marked sensory symptoms here than in an anterior poliomyelitis.

Dr. Percy Bolton said that he had seen quite a number of cases, the great majority having been apparently due to lesions in the spinal cord. Last year, however, he had seen one case in which the symptoms pointed to injury of the nerve roots in the foramina. This patient had fallen on his head, and had entered the hospital with tingling sensations in the distribution of the ulnar nerve. On flexing the head

on the trunk the tingling sensations in the arms could be developed, and this had led to the supposition that the nerve roots were involved.

#### PARALYSIS OF SERRATUS MAGNUS—GLIOSIS OF MID-DORSAL SEGMENTS.

Dr. William Hirsch presented a man who had come under his observation about two weeks ago. The patient stated that for two years past he had suffered from what he called "an itch on the abdomen." Examination had revealed the fact that in spite of the itching complained of there were no scratch marks visible. There was an area of disturbed sensation on the abdomen. Its upper border was formed by a horizontal line passing through the ensiform cartilage, and the lower border was formed by a horizontal line about six inches lower down. The lateral boundaries were the axillary lines. Further inquiry showed that there was no true pruritus, but a paresthesia. This area was extremely sensitive to heat and cold. Closer examination showed bilateral paralysis of the serratus magnus, as indicated by the wing-like projection of the scapulæ when the arms were held forward and horizontally. There was atrophy of the trapezius and of the latissimus dorsi. The patellar reflexes were normal. These muscular affections must have developed quite rapidly, as the man had been examined not long ago by a thoroughly competent physician in a hospital. He assumed that there was here a lesion in the posterior horns in the eighth or ninth dorsal segments—probably a neoplasm, a gliosis. This gliosis by occlusion of blood vessels and the consequent increased blood pressure might have caused the formation of a cavity. Another theory was that the primary gliosis had been too slight to cause any symptoms, but that by proliferation the effect observed had been produced.

Dr. R. G. Wiener said that this patient had been sent to him about a week ago for diagnosis. The man was a glazier by occupation, and anyone who was accustomed to work with the hands above the head was likely to suffer from paralysis of the serratus magnus. Careful examination of the affected area, when the patient's attention had been distracted, had led him to look upon the paresthesia as purely hysterical. The muscular paralysis seemed to him entirely peripheral.

Dr. Hirsch expressed great surprise at this diagnosis. There was only one case in literature of peripheral bilateral paralysis of the serratus magnus. Moreover, there were no sensory disturbances in the upper region. He could not reconcile the diagnosis of hysteria with the known pathology of the case presenting such symptoms. The affection of the other muscles made such a diagnosis, in his opinion, utterly impossible. The comparatively long duration of the case certainly did not exclude a gliosis.

#### OPHTHALMOPLAGIC MIGRAINE.

Dr. B. Onuf presented a young girl, who for the past five

years had suffered from attacks of pain in the right upper eyelid, associated with nausea and vomiting. Soon after the beginning of the attack the eyelid began to droop, and in the course of three days the eye was closed, and the eye appeared to be fixed. After remaining closed for about three days, the eye opened again, and the pain disappeared. Lately these attacks had been less frequent, there having been only about three in one year. Examination showed paresis of all the muscles supplied by the third and fourth nerves, but the rectus internus acted well. The attacks were preceded by constipation and chilliness. The case was looked upon as one of ophthalmoplegic migraine.

Dr. W. M. Leszynsky said that he would rather call the case one of recurring ocular palsy. He had seen one case in which there had been two attacks in one year. She had ultimately recovered completely.

Dr. Onuf replied that vomiting, periodic pain, and the associated tachycardia seemed to him to stamp the case as one of migraine in addition to the ocular palsy.

#### PARESTHETIC MERALGIA.

Dr. P. Meirowitz presented two men. The first was forty-eight years of age, and had first come to him on January 23 complaining of a gnawing sensation on the outer aspect of the thigh. There were also alternating sensations of heat and cold. Examination showed no objective changes in the appearance of the skin. Over the area, beginning three inches above the external condyle, and extending upward for some distance was anesthesia to touch, and immediately surrounding this was a region of tactile hyperesthesia. In portions of the affected region the strongest faradic current did not cause the slightest inconvenience.

The second patient was forty-seven years of age, and had been first seen on January 6, when he complained of a burning sensation on the outer side of the left thigh. The trouble had come on suddenly eight or ten days previously, and had been at first on the anterior aspect of the thigh. Occasionally the sensation was that of hot water boiling under the skin. Just above the knee was an area of anesthesia to cold. The affected area felt cooler to the touch.

Dr. C. L. Dana said that it had been very properly stated by Dr. Meirowitz that these cases were not at all rare. He had seen a similar condition affecting the calves of the legs on both sides. Probably paresthetic meralgias could be found in the distribution of most of the nerves.

#### INTRACRANIAL TUMOR.

Dr. Graeme M. Hammond reported a case of this kind. The only lesion discovered at autopsy had been an epithelioma

growing from the sella turcica. It measured  $7\frac{1}{2}$  by 2 by  $1\frac{1}{2}$  mm. The median line of the tumor corresponded very closely with the median line of the skull. It had compressed both optic nerves, the optic chiasm, part of both optic tracts and the third, fourth, fifth, and sixth nerves on both sides, both crura, the hypophysis, and the inner surface of both temporal lobes. Microscopical examination had not yet been made. In October, 1897, this woman, thirty-four years of age, had presented herself, and a diagnosis of encephalic tumor had been made. She had a good personal history. About fifteen years ago she had had a fall on the back of the head, but this had not been followed by any evidence of injury. Some years before coming under observation she had begun to suffer from a tic in the face, which had been relieved by extraction of several carious teeth. Shortly after this the articulation had become imperfect for a time, and soon innervation had become impaired. In August, 1897, she had suffered every morning from intense pain in the top of the head, and later on vomiting had been quite troublesome. In October, 1897, examination had shown the pupils to be regular and dilated, and responsive to accommodation. The eyeballs moved normally, but showed slight nystagmoid movements. She could not read fine print. Sensibility was normal. The ophthalmoscope revealed optic neuritis. During the next year the same symptoms had been present, but to these had been added severe headache, vertigo and a staggering gait. In September, 1899, weakness had been noticed in the left arm and leg, and after a time a well-marked hemiplegia had developed. In January the limbs were rigid, and the reflexes exaggerated. Sensibility was normal in all parts of the body, and smell, hearing and taste were normal. There was a well-marked double optic neuritis. The tumor was thought to be situated in the motor fibers of the right hemisphere. At the husband's request an operation had been performed, though no hope of a successful result had been entertained. The tumor had not been found, and the patient had never rallied from the operation. The remarkable feature was that such a large tumor, pressing on so many cranial nerves, should not have given rise to symptoms which would have made the location diagnosis reasonably certain. A search through the literature showed the reports of several cases in which tumors similarly situated had given rise to no focal symptoms.

Dr. Sachs said that he had had under observation for the past two years a case giving very much the same symptoms, and he had been impressed with the paucity of the usual symptoms of tumor of the brain. The only symptom added of late had been double anosmia.

## MYELITIS, WITH ESPECIAL REFERENCE TO THE TUBERCULOUS VARIETY.

Dr. Joseph Collins read a paper with this title. He stated that acute myelitis was relatively uncommon. The case reported was that of a woman, twenty years of age, who had been admitted to the City Hospital in 1898. Her illness had begun about three months before its fatal termination, the first thing noticed having been a redness and painful swelling of the second metacarpo-phalangeal articulation. The question arose whether these symptoms were really due to rheumatism, or to vasomotor changes. A few days later the right elbow had become painful and swollen, and in a few days more the other elbow, one ankle and one knee had been successively involved. According to the history she had been treated in Bellevue Hospital for rheumatism. On reaching the City Hospital she had had a number of large bed-sores of long-standing. She was pregnant at the time, and was soon after delivered of a child, which was now living. It was noted shortly afterward that the limbs were absolutely fixed, that there was urinary incontinence, but no sensory disturbances. The cranial nerves were not affected. About two weeks before death there had been rigors and elevations of temperature, indicative of septicemia.

The autopsy had been made twelve hours after death. The spinal canal contained pus up to the second lumbar vertebra, and from this point upward was a layer of greenish exudate, which reached to the brain and forward to the optic chiasm. The cord was very much softened between the sixth and ninth segments. The lungs were free from tuberculosis. Microscopical examination of the spinal cord showed the changes to be most marked in the eighth and ninth segments, and least marked in the cervical cord. From one end of the cord to the other the dura was the seat of productive inflammation, and this was true, to a less extent, of the pia. The sections made from the eighth dorsal segment showed the central area of necrotic tissue limited in the front by the commissure. One of the sections from this segment showed a few tubercle bacilli. No bacilli were found in the meningeal exudate. The changes in the ganglion cells were those of advanced diffuse chromatolysis. In many of the cells there was a complete disappearance of the chromatic granules. The cells most affected at these levels were those of the internal groups of the anterior horns. Sections from the first lumbar segment showed the thickening of the meninges and the inflammatory exudate, and also congestion of the blood vessels of the an-

terior horns. The changes in the chromatic granules and the achromatic substance were apparently secondary to the extensive changes in the blood vessels, and not dependent upon rheumatism or tuberculosis. Sections from the various lumbar segments stained by Nissl's method, showed variable disintegration and degeneration of the cells of the anterior horns. The processes of the cells showed a degeneration similar to that of the bodies. The so-called dislocation of the nucleus of the cell did not seem to him to indicate to any extent the pathological state to which the cell had been reduced. The changes in the cells of the cervical cord were similar to those in the lumbar region, though less pronounced. Many of the cells were large and stained deeply, while the achromatic substance in the cells was in good condition. The enormous thickening of the pia at this level was well shown in sections stained with hematoxylin. The cells of the anterior horns showed slight disintegration of the chromatic substance only. As the dorsal region was approached the cells of the anterior horns showed more marked degenerative changes. Here the remarkable feature was the relative preservation of the cells of Clarke's column. In conclusion, the speaker said that he did not believe that the pain in the extremities were the result of rheumatism but of tuberculosis.

Dr. Schlapp said that the first drawing exhibited showed a very large lesion, which seemed to him difficult to explain by the clinical history, particularly in view of the fact that there had been no sensory symptoms. He would like, therefore, to know whether it was not possible that this large lesion might have been the result of post-mortem change.

Dr. W. M. Leszynsky suggested that it was possible that the large bed sore over the sacrum might have had something to do with the septic process and the meningitis.

Dr. Collins replied that he had commented, in his paper, upon the point raised by Dr. Schlapp, and had stated that, in all probability, this necrotic area had been increased perhaps one-third by an artefact.

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- 28 DIE NERVENZELLE IN IHREN ANATOMISCHEN, PHYSIOLOGISCHEN UND PATHOLOGISCHEN BEZIEHUNGEN NACH DEN NEUESTEN UNTERSUCHUNGEN (The Anatomical, Physiological and Pathological Relations of the Nerve Cell as Studied by the Newer Methods). Ottone Barbacci (Centralblatt f. allg. Pathologie u. pathologische Anatomie, 10, 1899, Nos. 19, 20, 21, 22).

Attention is here directed to one of the most complete and valuable résumés of the recent work done on the nervous system. Barbacci's Zusammenfassendes Referate should certainly be in the library of neurologists interested in the advancements of the newer nerve cytology.

JELLIFFE.

## THE PHILADELPHIA NEUROLOGICAL SOCIETY.

December 18, 1899.

The President, Dr. James Hendrie Lloyd, in the chair.

Dr. Wharton Sinkler reported a case of acute poliomyelitis anterior in youth seventeen years of age. (See p. 205).

Dr. William G. Spiller said that he had examined specimens from three cases of anterior poliomyelitis, and in two of these cases death had occurred a few days after the commencement of the disease. Last spring Dr. Sherman, of Buffalo, sent him the spinal cord and parts of the brain from a case which had been regarded as one of Landry's paralysis. They showed the lesions which have been found a number of times in anterior poliomyelitis. There was intense perivascular round-cell infiltration and the motor cells of the cord were almost entirely destroyed. No nerves from this case were obtained. The changes were not limited to the anterior horns, although much more intense there, and perivascular infiltration was found in the white columns and the meninges were affected.

Dr. Spiller said he had seen a few cases of anterior poliomyelitis in which the paralysis was limited to some of the muscles of the leg below the knee, and in these cases the knee-jerk was preserved.

It does not follow because some pain is present in these cases with symptoms of poliomyelitis, that the condition is one of neuritis. As the inflammation is not strictly limited to the anterior horns, it is easy to understand that some pain may occur, although it does out a wheel-chair or crutches.

Dr. Charles W. Burr said that six or seven years ago Dr. Sinkler had had a case of acute infantile spinal palsy in which death occurred in the acute stage. Dr. Burr had examined the spinal cord and found that the lesions were not confined to the anterior gray matter, although that region was the seat of the most marked disturbance. There was an acute transverse myelitis affecting the anterior cells in the lumbar cord, and causing almost their entire destruction, and the whole white matter was involved to a greater or less degree.

Dr. A. A. Eshner said that five years ago he had observed a case similar to the one reported, in a man thirty-three years of age, the only point of etiological significance being the occurrence of gonorrhea fifteen years previously. In this man the first notable symptoms were some difficulty in the expulsion of urine and slight loss of power in the left lower extremity which later became more marked. There were enfeeblement of the knee-jerk and wasting on the affected side, but no sensory symptoms. This man is now unable to get about without a wheeled chair or crutches.

With regard to etiology it seems probable that anterior poliomyelitis is not due to a single specific cause, but may be brought about by a variety of conditions. The disease may be the expression of a variety of infections, with predominant localization in the anterior horns of the spinal cord.

The differentiation between peripheral neuritis, particularly of the motor variety, and anterior poliomyelitis is at times extremely difficult. Viewing the lower motor segment as one continuous whole, it is easy to understand that in certain instances the motor cells of the cord may suffer most, or exclusively, while in other instances the nerve fibers may suffer especially; or there may be simultaneous in-



volvement of the motor cells and the afferent axones. In this way phenomena may occur that are more peculiarly spinal, or peculiarly peripheral, or finally, a mixture of both. Ordinary multiple neuritis is commonly the manifestation of a general toxic process, so that one may reasonably expect involvement of both sensory and motor nerves.

Dr. James Hendrie Lloyd remarked that Dr. Sinkler's statement that neuritis may have been present in this case recalled a case at the Philadelphia Hospital which he had reported some years ago. The patient was a ship carpenter, who, when crossing the equator, and being much overheated, had permitted himself to be drenched with buckets of water taken from the sea, which, of course, could not have been very cold. This bath sufficed, however, to bring on paralytic symptoms that completely invalidated him in the course of a few days. He was taken to a hospital in Rio, and in the fall of that year came to Philadelphia. When he arrived at the Philadelphia Hospital, he was in a condition of well-marked locomotor ataxia. There was also a history of syphilis. The abrupt onset of paralytic symptoms following sudden checking of the perspiration suggested that the case had been originally complicated with multiple neuritis, which gradually recovered, leaving the central lesion permanent.

Dr. Wharton Sinkler said with regard to pain, that there was no more constant symptom in poliomyelitis at the onset, than pain. It is of a totally different character from the pain of peripheral neuritis. It is more of a myalgia, and is increased by muscular movements. There is no true hyperesthesia. That character of pain is sufficient to establish the differentiation between this disease and peripheral neuritis. The fact that the loss of power so promptly receded from the right leg in the patient shown by him, was another characteristic of poliomyelitis and quite different from what is seen in multiple neuritis.

Dr. James Hendrie Lloyd exhibited a patient with unilateral motor disturbance.

The patient was a white woman aged 22 years. The family history was negative. When 12 years of age, she had attacks in which she felt as though she had a lump in her throat. Menstruation began at the age of 18 years, but had been irregular, there being sometimes one year between the periods.

Movements similar to those which she now presents began about five years ago, and have occurred at intervals of two or three weeks ever since. The present attack began nine weeks ago.

The movements consist in a more or less purposive extension and drawing up of the left leg and a pounding of the bed or limb with the left hand. These movements continue during the day and disappear when she sleeps.

There is no area of anesthesia, and the heart and lungs are apparently normal. (This case will be made the text for a paper to be read at a future meeting of the society.)

Dr. Charles K. Mills thought that there was little doubt that this was a case of what might be termed hysterical rhythmic chorea. Nearly twenty years ago he had seen a very similar case and had shown it to the County Medical Society, where he hypnotized the patient. In this case the movements were on both sides. The patient did not

improve under treatment. After some years she was sent to Lourdes, the famous religious resort, and there was cured.

Dr. F. S. Pearce remarked that the rhythmic condition reminded him of a case which he had seen in Dr. S. Weir Mitchell's clinic. The patient was a woman who had rhythmic up-and-down spasmodic movements of the larynx, occurring eighty or ninety times per minute. It was supposed to be due to the "use of a pessary." Dilatation of the uterus was done and various other suggestions made, but without any effect. Dr. Mitchell considered the case as hysterical.

Dr. A. A. Eshner referred to a case reported in 1876, by Dr. Weir Mitchell as one of pendulum-spasm, in which a rhythmic movement not unlike that exhibited by Dr. Lloyd's patient has been present in all the intervening years, except for a brief period recently when the movement yielded to hypnotic suggestion on the part of Dr. J. Madison Taylor. Subsequently, however, recurrence took place as a result of some emotional excitement. Renewed treatment was again followed by a cessation of the movement, but the man evidently came to the conclusion that he led a more successful life with the movements, so that he has declined further treatment. The movements occurred in the erect posture and ceased in recumbency, and when the hand was raised above the head.

Dr. Wharton Sinkler said that the movements in this case were characteristic of hysterical rhythmical chorea. He had seen a number of such cases. Seven years ago, he had under treatment a young woman with almost identical movements of the right upper extremity which had lasted for three or four months. She was cured in about a month and remained well. Three or four days ago she came to Dr. Sinkler's office bringing her child twenty-one months old, rachitic, with a tendency to hydrocephalus and a very distinct rotary movement of the head, but without nystagmus. This he considered rather curious in view of the previous history of the mother.

Dr. James Hendrie Lloyd said that while his first impression was that this was a hysterical case, yet he was not sure of it until after he had studied it with some care. It was unlike any motor disorder of hysteria that he had seen or read about. It was not inco-ordination, and it was not tremor. It was unilateral, which is rather in accord with hysterical phenomena. It had not been possible to determine any other hysterical manifestations in the case. He thought that the case might properly be called one of hysterical choreaform movement.

Dr. Charles K. Mills described a case of unilateral progressive ascending paralysis, probably representing a new form of degenerative disease. (See p. 195).

Dr. Wharton Sinkler said that he had examined this patient very carefully. The diagnosis in his mind lay between two conditions: either a focal destructive lesion of the posterior part of the internal capsule or thalamus, or inflammation of the spinal nerve roots. He thought that the former might be the case, from the peculiar inco-ordination of the right hand. This hand could not be used unless the patient watched it. The absence of cerebral symptoms, the absence of fundus oculi changes and of convulsions, led the speaker to be more inclined to the view that this patient might have involvement of the spinal nerves at their points of emergence from the spinal canal. This seemed plausible from the hyperesthesia which was a conspicuous symptom, and the attack of herpes zoster preceding or accompanying the motor symptoms.

Dr. Charles K. Mills regarded the sensory symptoms which had been present in his case rather as epiphenomena. The man had not-

able weakness in the right leg for several months before he had this attack of pain in the gluteal region and hips. He did not consider this an essential feature of the disease. The case did not represent clearly any form of disease described in the books.

#### ACUTE INTERNAL HYDROCEPHALUS.

Dr. Chas. W. Burr and Dr. D. J. McCarthy reported the following case: W. S., a fireman by occupation, was admitted to the service of Dr. Burr at the Philadelphia Hospital, with intense pain in the head, and rigidity of the neck, and later had delirium and unconsciousness. The temperature was elevated, the tongue coated, and marked constipation was present. This condition lasted for three weeks, and was marked by the vacillating, intermittent character of the symptoms. At the end of three weeks the temperature fell to normal, and a condition resembling very much the initial stage of general paralysis of the insane developed. About the middle of the sixth week the temperature went up to 103 degrees, and the patient presented the same symptoms as were present on admission. This condition lasted five days, and was followed by a period of purely psychical phenomena. Another exacerbation of the febrile symptoms occurred, and the patient died in a condition of low muttering delirium. At the autopsy internal hydrocephalus, with marked edema of the cord was found. Microscopical examination revealed widespread inflammatory changes, affecting the ependyma and subependymal tissues; acute and chronic inflammatory degenerative changes in the choroid plexus, and degeneration of both auditory nerves. No changes were present in the cord to explain the disappearance and return of the knee-jerk which occurred during the course of the disease. Experimental injection of toxic substances and acid irritants into the ventricles of animals revealed interesting reactive changes in the ependyma and subependymal tissues, resembling the changes described above, and in so far supplied a toxemic origin of the internal hydrocephalus.

Dr. William G. Spiller had seen this case in Dr. Burr's service and thought that at that time it presented much the appearance of meningitis. He had also examined the interesting specimens. It has been held by many that a poison may exist in the cerebro-spinal fluid and act upon the brain and cord, producing inflammation, but he was not aware that this had been demonstrated experimentally in the way that it had been done by Drs. Burr and McCarthy. Ependymal granulations are not very uncommon in cases of parietic dementia and cerebral syphilis. He had seen Argyll-Robertson pupil in syphilis. This condition in syphilis has been explained as the result of ependymal granulations and proliferation of the subependymal tissue of the aqueduct of Sylvius. It is possible that the polioencephalitis superior that occurs in alcoholic subjects may be produced in the same way.

but this is doubtful, as it is strange that the irritation is not manifested also in the area around the fourth ventricle.

In some of the specimens of the case reported there were granulations which resembled, very closely, some which Dr. Spiller had seen in preparations made by Dr. Sottas, of Paris, in his study of cerebro-spinal syphilis.

It has been thought that in some cases where disease rapidly extends up the spinal cord, that the poison has been transmitted through the central canal. That may be possible, especially in cases of hydro-myelia.

In the case of Drs. Burr and McCarthy, the spinal cord was said to be exceedingly edematous, so much so that it bulged very greatly. A similar condition had been recently reported in a case of brain tumor, and the loss of knee-jerk that had occurred was explained by this edema of the cord, producing degeneration of the posterior roots. In the case of Drs. Burr and McCarthy, no degeneration of the posterior roots was observed.

The loss of hearing was interesting. There was distinct degeneration of the acoustic nerves, and possibly this degeneration was due to the pressure of the fluid in the fourth ventricle.

Dr. Joseph Sailer reported a case of motor and sensory disturbance of obscure nature.

Dr. William G. Spiller remarked that the disturbance of sensation and motion on the same side of the body made the diagnosis of a spinal lesion doubtful.

Dr. Charles W. Burr asked Dr. Spiller whether it had yet been proven where the nerves of pain and heat and cold sensation cross in the cord.

Dr. Spiller replied that the course of sensory fibers has not been so well worked out as that of motor fibers. The view held by many investigators is, that the fibers of so-called muscular sense or deep sensation, ascend in the posterior columns; the fibers from the lower limbs in the columns of Goll, and those from the upper limbs in the columns of Burdach. Dr. Spiller said he had never found degeneration in the lateral columns from lesions of the posterior roots. This has been seen in experimental work and is probably due to traumatism of the antero-lateral columns. Considerable evidence is found for the view that the tactile fibers ascend in the posterior columns, and a case of syringomyelia reported by Dr. Dercum and himself, was of value in this connection. Some hold that the tactile fibers ascend in the anterior-lateral columns after decussating. It is probable that the fibers of temperature and pain sense decussate after entering the cord—as was evident in the case of syringomyelia limited to one posterior horn in the cervical region, reported by Dr. Dercum and himself—and that the posterior root fibers end in the gray matter, and new neurons commence in that part. In Brown-Séquard paralysis motion is affected on one side and pain and temperature sense on the other, but in many cases tactile sense is intact on both sides.

Dr. F. S. Pearce said that the paresis of the face which he understood had occurred at one time, with the nystagmus, would indicate some involvement of the cerebrum. The idea of cerebro-spinal vasomotor change with serous effusion would correspond with the symptoms of the case, and would also, he thought, agree with the rapid convalescence.

## Periscope.

### CLINICAL NEUROLOGY.

29. TABÈS ET TRAUMATISME (Tabes and Traumatism). Donadieu-Lavit (Archives provinciales de Médecine, I, 1899, p. 205).

Donadieu-Lavit reports a case of tabes in which the symptoms were much exaggerated, after a fracture of both bones of the right leg near the ankle. The patient, a man 43 years old, was alcoholic and syphilitic. He walked badly, but he could walk with the aid of his sight. Because of his ataxia he fell and sustained the above mentioned fracture, the tibia projecting from a wound. The leg was put up properly in a plaster splint for three months and was then well united and healed. Although his muscles seemed strong, on attempting to rise and walk he fell helpless to the floor. The ataxia was much increased in the right leg, which was also larger than the left and edematous. The callus was very abundant, and slightly painful on pressure, and there was distension of the synovial sac, so that the ankle looked like a Charcot joint. The sense of position was entirely lost in the right leg. There was anesthesia in the foot, at the level of the callus, and over the buttocks. Sensation was much delayed in the right leg. Both buttocks were somewhat atrophied, the right more than the left, but the legs were not. The knee jerks were both absent, the plantar reflex was lost on the right only. The lightning pains were more severe than before the fall, especially in the right leg. If the patient wished to walk it was necessary for him to see his right foot advanced and placed firmly upon the ground. An elastic stocking extending well up the thigh gave great support to the right ankle and leg. The patient was given mercurial inunctions, massage, gymnastics, hot baths, and the symptoms gradually diminished, or disappeared, until he could walk as well as before the fall. His ataxia, the anesthesia, and the Romberg sign have diminished, and the atrophy of his buttocks has disappeared. The arthritis, the callus and the edema have also become much less.

Donadieu-Lavit concludes that the immobilization in the dorsal position was the direct cause of the sensory, motor and trophic disturbances and that they exaggerated the ataxic weakness so as to confine the patient to bed.

BONAR.

30. NEURALGIA EPIDEMICA (LOCALISÉ) (Epidemic Neuralgia). Wille (Münchener medicinischer Wochenschrift, 1899, Nos. 33, 34, and 35).

The author, a physician practicing in a country district of Swabia, having encountered within three months (August 10, 1898, to November 10, 1898), and in a comparatively limited territory, a number of cases of neuralgia, enormously out of proportion to anything previously met with, in an experience of twenty years, was led to make a study of the epidemic.

He presents short clinical histories of forty-nine cases of his own, and adding to them sixty-three cases from the practice of neighboring physicians, tabulates the whole number, and considers them in relation, the age of the patients, the nerves affected, the region of the district in which they occurred, the date, etc.

He next enters into a discussion of the physical features of the country, and of the meteorological conditions prevailing at the time.

The article presents a long and careful study of the subject, but the author does not seem to have arrived at any definite conclusion as to an infectious agent.

ALLEN.

31. UEBER DIAGNOSE UND ERFOLGREICHE CHIRURGISCHE BEHANDLUNG VON GESCHWÜLSTEN DER RÜCKENMARKSHÄUTE (Concerning the Diagnosis and Successful Surgical Treatment of Tumors of the Spinal Membranes). F. Schultze (*Deutsche Zeitschrift für Nervenheilkunde*, 16, 1899, p. 114).

Schultze reports two cases of spinal tumor with removal of the growth. In the first case after a paralysis lasting 17 months, which had been complete for 13½ months, and was associated with considerable disturbance of sensation, improvement was so great that one year after the operation the patient could walk short distances without support, and longer distances if assisted. The tumor was situated at about the fifth, sixth and seventh thoracic vertebræ. In the second case an almost complete motor spastic paralysis, with some disturbance of sensation, almost disappeared in five or six months after the operation. The tumor in this case was a fibrosarcoma and was situated at about the seventh thoracic vertebra. In Bruns' statistics of spinal tumor, 20 cases, with improvement or recovery after operation in 6, are reported. In the third case reported by Schultze a fibroma at the foramen magnum was found at the necropsy. Absence of pain and of rigidity of the neck was observed for a long time, and the extremities were rigid. This tumor caused paresthesia and disturbance of motion in the left lower limb before it caused symptoms in the left upper limb, and closed the right side of the foramen magnum. Schultze believed that the case showed that in the upper cervical region the motor fibres for the leg are more peripheral than those for the arm. A fourth case reported by Schultze was one of tumor of the cord, extending from the conus high into the thoracic region. It appeared to be a glioma.

SPILLER.

32. ON THE TEMPERATURE IN CASES OF APOPLEXY, AND ON THE OCCURRENCE (1) OF OEDEMA AND (2) LOSS OF THE KNEE-JERK IN THE PARALYSED LIMBS IN HEMIPLEGIA. J. Michell Clarke (*The Bristol Medico-Chirurgical Journal*, Vol. 17, June, 1899, p. 972).

In cases of cerebral hemorrhage there is an initial fall of the temperature of the body. In rapidly fatal cases this subnormal temperature continues, though in some which live for some hours, the temperature may afterwards rise to a high level. In cases that prove fatal after a few days, the initial fall is succeeded by a stationary period of return to, or near, normal, ending with a rise of temperature before death. In cases which recover, the temperature soon returns to normal, or to slightly below normal, after an initial fall and rise. On the other hand, in softening due to thrombosis, there is only a very slight initial fall, if any. The temperature usually rises, and this is followed by oscillations, sometimes marked.

Dana (*Post-Graduate*, 1896, XI, 316) has called attention to the fact that in cases of hemiplegia, due to cerebral hemorrhage, the temperature of the paralyzed side is higher than that of the sound side, and that this difference in temperature is not present in acute cerebral softening from thrombosis or embolism. This fact the writer thinks may be of practical use in making a differential diagnosis between softening due to embolism or thrombosis, and cerebral hemorrhage. Dana also says that he has never found any disturbance of temperature in hemiplegia due to embolism.

The writer reports some cases with their temperature charts. He finds that the temperature on the affected side in two cases of hemorrhage varied from one-half to two degrees higher than that on the sound side. In one case of hemorrhage into the pons, the temperature was the same on both sides of the body and was persistently subnormal, but this case had numerous convulsions and all four limbs were paralyzed. In one case in which the temperature of the affected side was one degree higher than that of the other side, thirty minutes after death the temperature was the same on both sides, that is, the temperature of the paralyzed side ceased to rise, while that of the sound side rose one degree after death. While there was a rise of temperature in another case, with a difference most of the time of two degrees between the two sides, just before death the temperature of the two sides fell to the same level. This was a case of white softening and the temperature chart shows that, while the temperature difference between the two sides resembled that of hemorrhage, it was more irregular, and nearly corresponded on the two sides.

The writer cannot explain the fact of the lower temperature on the side of the lesion, unless it is due to some controlling influence of the sound hemisphere upon the temperature of the opposite side of the body. The fact of the temperature of the sound side rising one degree after death, in one case, is interesting in this connection.

He also cites four other cases which recovered, illustrating respectively the temperature-difference between the two sides in hemorrhage; a similar, but slighter difference in cerebral syphilis, due probably to occlusion of a vessel; the subnormal temperature which may obtain for some time in hemiplegia; and a want of correspondence between the surface temperature and that in the axilla. In two cases there was edema, accompanied by pain and hyperesthesia of the affected side, although there was no reason to suspect kidney disease. In one of these cases and in two others the knee-jerk of the paralyzed side was absent. Hemianesthesia of the affected side was also present in several cases.

The writer cannot explain this loss of knee-jerk in the paralyzed leg, which is contrary to what usually obtains. BONAR.

33. *UEBER GEHÄUFTES AUFTRETEN UND ÜBER DIE AETIOLOGIE DER POLIOMYELITIS ANTERIOR ACUTA INFANTUM* (Concerning Endemic Appearance and Etiology of Acute Anterior Poliomyelitis). Siegmund Auerbach (Jahrbuch f. Kinderheilk., 50, 1899, No. 1).

At the Polyclinic at Frankfurt there occurred in the seven months from May to December, 1898, 15 cases of poliomyelitis, while in the 5 years previously only 11 cases were recorded. In one of the cases, a facial paralysis, nuclear in origin, was present. From a study of the literature of the subject, and of lumbar puncture in cerebrospinal meningitis, the writer comes to the conclusion that the difference between epidemic anterior poliomyelitis, cerebrospinal meningitis, sporadic or epidemic, and encephalitis, is not one of pathogenesis, but merely that of selection and degree of intensity of action of the same micro-organism, *i. e.*, the Weichselbaum-Jäger meningococcus. While the bacteriological investigation of the spinal fluid was not made in any of the 15 cases reported, lumbar puncture and the study of the fluid is urged from a therapeutic and diagnostic standpoint.

McCARTHY.

34. ZUR DIFFERENTIALDIAGNOSE DER MULTIPLEN SKLEROSE (Differential Diagnosis of Multiple Sclerosis). D. Gerhardt (Deutsche Zeitschrift für Nervenheilkunde, Vol. 15, 1899, p. 458).

Gerhardt reports a case in which he observed intention tremor, spasticity of the limbs, scanning speech, apoplectic attacks—in short, the clinical picture of disseminated sclerosis without nystagmus. The intelligence began to fail, dementia became pronounced, and muscular twitchings and grinding of the teeth were noticed. The necropsy showed macroscopically increased consistency of the brain and spinal cord, which was believed to be due to diffuse sclerosis, but microscopically this sclerosis was observed only in the cord. The case bore some resemblance also to one of general paralysis. Atrophy of the cerebrum, especially of the frontal lobes, slight chronic leptomeningitis, accumulations of cells and pigment in the vascular sheaths, destruction of tangential fibers—these were very like the findings of general paralysis, but there was no ependymitis granulosa. The dementia was different from that of general paralysis; it increased with the decrease of the apoplectic attacks, was not associated with ideas of grandeur, and was like the dementia of cerebral hemorrhage or softening. The mental disturbance occurs early in parietic dementia, but in Gerhardt's case it came late in the disease, and after the symptoms of multiple sclerosis were seen. Diffuse sclerosis should be thought of when the symptoms of multiple sclerosis are present without nystagmus, when mental failure becomes more and more prominent, when the scanning speech becomes more like that of general paralysis, and when muscular tremor and grinding of the teeth occur. This diffuse sclerosis cannot be separated clinically or histologically from Strümpell's pseudo-sclerosis.

Small multiple foci of softening as well as diffuse sclerosis can cause the clinical appearances of disseminated sclerosis, and a case of the kind is reported by Gerhardt. Usually multiple foci of softening cause bulbar symptoms, and the disease is more common later in life, while disseminated sclerosis is more common in early life. SPILLER.

35. LES DIFFÉRENTES FORMES DE PARAPLÉGIE DUES À LA COMPRESSION DE LA MOELLE ÉPINIÈRE ET LEUR PHYSIOLOGIE PATHOLOGIQUE (The General Symptoms of Paraplegia Due to Compression of the Spinal Cord, and its Pathological Physiology). A. van Gehuchten (La Presse médicale, 1899, p. 218).

In this exceedingly valuable article, van Gehuchten undertakes to analyze and explain the various symptoms produced by complete destruction of a transverse segment of the cord. He divides a typical case of paraplegia into four degrees. In the first there is spasmodic paraplegia, exaggeration of the reflexes, but no sensory disturbances. In the second, flaccid paraplegia, loss of reflexes, but still no sensory disturbances. In the third, the motor symptoms are the same as those in the second degree, but there is the syringomyelic dissociation of sensation; and in the fourth, the motor symptoms are still unchanged, but there is complete anesthesia. Between these types there are, however, an infinite number of intermediate stages. Naturally, all the four forms usually occur successively in the same patient, providing the cause of the lesion is persistent and progressive. Therefore, van Gehuchten has prepared a second and longer table in which he includes the most characteristic of the transitional forms. These, however, are essentially characterized by partial instead of complete sensory disturbances. In a brief review of the anatomy of the tracts of the cord, he includes among them the motor fibers, the antero-lateral pyramidal



tracts, the cerebello-spinal tracts, uniting the cerebellum with the anterior cornua of the cord and motor fibers belonging to the posterior longitudinal fascicle that arises from a group of cells just beneath the oculo motor nucleus. He also believes that descending fibers pass into the spinal cord from the nucleus rubrum and the nucleus of Flechsig. Of all these fibers the most important are the cortico-spinal group, and in the first stage of compression they alone are involved and give rise to the syndrome of spasmodic paraplegia. As the pressure increases, all the descending fibers become involved, paraplegia becomes flaccid, and the reflexes are lost. Further compression produced alterations in sensation, involving first the fibers conveying pain and heat sensation which pass up through the gray matter of the cord, and ultimately the fibers of muscle sense which occupy the columns of Goll and Burdach. Sometimes there is isolated disturbance of pain sensation while heat sensation remains normal, or the area of disturbance of pain sensation is more disturbed than the area of heat sensation. Van Gehuchten questions whether the same fibers convey both forms. It is curious that although the fibers of the posterior columns in the cerebellar tract are most resistant to compression, they are most susceptible to syphilitic disease. There are many exceptions to these general rules and several cases are quoted, mostly from the literature, in which either the sensory disturbances occurred first, or there was flaccid paralysis with exaggeration of the reflexes. In a few rare cases there are motor disturbances with exaggeration of one or other forms of sensation; that is to say, some form of hyperesthesia. This is probably caused by the zone of hyperesthesia described in Brown-Sequard's paralysis. This hyperesthesia has been explained by Martinotti, as a result of the lesion of a narrow band of fibers that is situated in the angle between the anterior and posterior cornua. In all cases of total transverse destruction of the cord, hyperesthesia is absent. SAILER.

36. DES NÉURALGIES ET TICS DE LA FACE CONSIDÉRÉS DANS LEURS RAPPORTS AVEC UN ÉTAT PATHOLOGIQUE DES VOIES LACRYMALES (The Neuralgias and Tics of the Face Considered in Their Relation with a Pathologic Condition of the Lachrymal Ducts). P. Bettremieux (Archives provinciales de médecine, Vol. I, No. 10, October, 1899, p. 703).

The writer believes that there is a pathogenic relation between the neuralgias and tics of the face and diseased conditions of the lachrymo-nasal mucous membrane. He reports nine cases, four of neuralgia, three of tic convulsif, and two of tic douloureux, in which the results of his treatment tend to uphold his opinion. Of these nine cases, a marked improvement, or cure, was obtained in six, no result was obtained in one, and two were lost sight of. In nearly all of these cases there was increased lachrymation on the affected side, which had not been noticed by the patient. The treatment consisted of either simple catheterization of the lachrymal duct, catheterization with injections of nitrate of silver solution or some other antiseptic, or the passage of sounds in the duct followed by injections. There was almost immediate relief from pain in those cases in which it had been present after one treatment, and convulsive movements were diminished in severity. It seemed in some cases to be necessary to keep up the treatment for some time, or the trouble would return. Those cases which were not cured enjoyed a longer respite between the attacks, which also were not so severe as formerly. Bettremieux concludes that the lachrymo-nasal mucous membrane, if diseased, may be the seat of neuralgias and tics of the face, and that this lesion may be superficial,

without any accompanying lachrymation, so that the relation between the cause and effect is not apparent. Such cases ought to be examined thoroughly. He thinks that neuralgias and tics of the face should be considered as pathologic reflexes, and that the origin of these reflexes should be sought for, especially in the region of the lachrymal ducts, before having recourse to surgery which only interrupts the reflex circuit.

BONAR.

37. OPTHALMOPLÉGIA INTERNA BEI MIGRAINE OPTHALMOPLÉGIQUE (Internal Ophthalmoplegia in Ophthalmoplegic Migraine). E. Troemner (Centralblatt für Nervenheilkunde und Psychiatrie, Oct., 1899, p. 577).

A woman of thirty-eight years of age had headaches since her twenty-first year, with the exception of a period of five years. The mother and sister of the patient also had headache. The patient's pain was sometimes on the right side of the head and sometimes on the left; it began almost every day in the morning, and was often associated with redness and swelling of the cheek and ceased after or during a long sleep. The headache, which had almost disappeared during a period of five years, returned after a fright caused by the sudden death of the patient's husband. On the second or third day after the pain again began the woman noticed that the vision of the right eye was impaired and that the right pupil was very large. Examination showed that the right pupil did not react to light or in convergence and reacted very imperfectly in accommodation. After three or four months considerable improvement in the ophthalmoplegia occurred. No other case of internal ophthalmoplegia associated with migraine seems to have been reported.

SPILLER.

38. UEBER PERONEUSLÄHMUNG BEI TABES (Concerning Paralysis of the Peroneal Nerve in Tabes). Finkelnburg (Monatsschrift für Psychiatrie und Neurologie, Vol. 6, No. 4, Oct. 1899, p. 286).

Peroneal paralysis has occasionally been seen in cases of tabes dorsalis. Finkelnburg reports two cases in which the paralysis in the distribution of the peroneal nerve resulted from pressure, therein differing from the other cases of tabetic peroneal paralysis in the literature. In the first case the pressure was caused by the position of the legs maintained for a long time, but alcoholism and tabes rendered the peroneal nerve more liable to injury from slight pressure. In the second case an arthropathy of the knee caused pressure on the peroneal nerve.

SPILLER.

## PATHOLOGY.

39. UEBER DAS GONOCOCCENTOXIN UND SEINE WIRKUNG AUF DAS NERVENSYSTEM (On the Gonococcus Toxine and Its Action on the Nervous System). Maltschauoff (Münchener medicinische Wochenschrift, 46, 1899, No. 31, p. 1013).

The author describes the changes found in the nervous system after the action of gonococcus toxine; in white mice, guinea pigs and rabbits.

As a culture medium for the gonococcus he used peptone agar, with hydrocele fluid, while for toxine production, the organism was allowed to grow in a mixture of equal parts of bouillon and hydrocele fluid. Cultures, 20 to 25 days old, in which the cocci had been killed by heating at 70° C. for 15 minutes, but which were used unfiltered, were found by the author to be most toxic.

White mice which had received injection of 0.5 to 2.0 gm. invariably died in from 12 to 28 hours, with symptoms of acute ascending paralysis. The nerve cells, especially those of the anterior horns of the spinal cord, showed acute degeneration, with chromatolysis, alteration in position of the nucleus, vacuolization, etc. In the brain hyperemia and small hemorrhages were found, but the peripheral nerves and muscles were unaltered. Guinea pigs proved quite susceptible, and after the injection into the peritoneal cavity of 10.0-15.0 gm., died in from 1 to 5 days, showing progressive emaciation and weakness. Rabbits died in convulsions 4 to 6 hours after the intravenous injection of 10.0 gm. or more of the toxine.

In both rabbits and guinea pigs it was found that a condition of chronic intoxication could be produced by repeated injections of less than the lethal dose. In both animals paresis or paralysis of the hind legs usually came on in from 1 month to 6 weeks. His conclusions are:

In acute intoxication the changes consisted in alteration (chromatolysis, excentric position of the nucleus and vacuolization) of the nerve cells, especially of those of the anterior horns, to a less extent of those of the intervertebral ganglia, least of those of the nuclei of the medulla and of the cortex.

In chronic intoxication, degenerative neuritis was most common, and sometimes degeneration of the posterior roots and of the posterior columns of the cord was observed.

ALLEN.

40. STUDIEN ZUR PATHOLOGIE UND PATHOLOGISCHEN ANATOMIE DER RÜCKENMARKSCOMPRESSION BEI WIRBELCARIES (Studies on the Pathology and Pathological Anatomy of Compression of the Spinal Cord in Vertebral Caries). Alfred Fickler (Deutsche Zeitschrift für Nervenheilkunde, 16, 1899, p. 1).

Fickler has had a very rich material at his disposal for a study of vertebral caries—20 cases in all, 19 of which were with necropsy. Some of his more important statements are: Tuberculosis of the spine is almost always secondary, and usually the lungs are the primary seat of infection, although the lymph glands are occasionally first affected, and more rarely other organs. Trauma seems to be of some importance in the development of the disease, although a cause is not usually found. The disease seems to appear somewhat more frequently after middle life, as in more than 50 per cent. of Fickler's cases the patients were over 50 years old. The first symptom is usually dull pain at the affected vertebræ, and is increased by bending over, but in children the pain is not infrequently absent, and in adults also it may be unimportant. Rigidity of the spine is another early symptom. Pain on pressure over the spine is absent in about half of the cases. Root symptoms, usually sensory, are among the earliest symptoms of the disease, and the pain is continuous, although it may occur periodically. The first symptoms of compression of the spinal cord are disturbances of motor innervation, such as fatigue, ataxia, and paresis. Paraplegia may occur suddenly, and when it does it is due to rupture of the abscess into the vertebral canal or to the giving away of carious vertebræ. One of the earliest symptoms of compression of the spinal cord is constipation. Unfortunately, Fickler has had no experience in operation in cases of Pott's disease. The most common cause of compression of the spinal cord is the extension of the tuberculous process to the epidural tissue.

A great many histological details are given by Fickler, among which the most interesting is the description of fibers within the cord,

believed by him to be newly-formed nerve fibers. From this finding he concludes that the nerve fibers of the spinal cord are capable of regeneration with complete restoration of function, provided the vascular system is intact. The recovery of function after spinal compression, he believes, depends upon the restoration of the normal lymph circulation, upon the formation of medullary sheaths about persisting axis cylinders, and upon the regeneration of nerve fibers. SPILLER.

41. ZUR PATHOLOGISCHEN ANATOMIE UND BACTERIOLOGIE DES DELIRIUM ACUTUM (The Pathological Anatomy and Bacteriology of Acute Delirium). A. D. Kazowsky (Centralbl. f. allg. Path. u. path. Anat., 10, 1899, p. 489).

After a brief summary of the important literature upon the subject, particularly that of Russian authors, two cases are reported in which it was possible to make extensive histological investigations, and in one of them also bacteriological studies. The first patient, a man, was brought to the hospital in a state of intense excitement, with destructive tendencies, and fever. There were hallucinations of sight, hyperesthesia, refusal of food, and occasionally vomiting. The patient did not sleep, became weak, and died on the sixth day of his residence in the hospital. At the autopsy, aside from hyperemia of the central nervous system and of the viscera, there was a slight tumor found upon the anterior vermiform process of the cerebellum. Microscopically, there was found distention of the blood vessels, peri-vascular and cellular accumulation, various alterations in the nerve cells and the nerve fibers, proliferation of the neuroglia, distended spaces around the ganglion cells in which cellular elements were found; the nerve cells showed alteration of the nuclei, and disappearance or dislocation of the nucleoli. In addition many of the cells contained a considerable quantity of yellowish pigment that resisted all reagents and stained only with osmic acid. Similar collections of pigment were found in the blood vessels. This pigmentary infiltration of the ganglion cells cannot be looked upon as abnormal, excepting in the areas in which there was some softening, where the cells contained considerably more pigment than in the other situations. No cultures were made from the tissue, but microscopically micrococci occasionally arranged in chains were discovered. The second patient, a soldier, was brought to the hospital in a state of extreme depression. He soon became excited, destructive, had fever, developed swelling in both parotid glands, had extreme exhaustion, and died. The whole period of the acute process lasted fifteen days. At the autopsy there was hyperemia of the brain, enlarged spleen, cloudy swelling and perhaps necrosis of the liver, and also some punctiform hemorrhages in the mucous membranes. Cultures from the cerebro-spinal fluid, the heart, blood and the spleen showed the presence of staphylococcus pyogenes aureus. Microscopically the features were similar to those in the other case. Pathologically, these two cases are identical; clinically, one represents an idiopathic acute delirium, the other an acute exacerbation of a pre-existing psychosis. Both are probably due to infection with pyogenic cocci. It appears from the study of these cases as well as from a consideration of the literature that we are not justified in considering acute delirium as an independent disease. It rather represents an acute infection, particularly localized in the central nervous system. It is difficult to explain, however, the route by which the micro-organisms reach the brain, but probably there is an infection of mild form that becomes first generalized and then localized in a brain already weakened by some other cause. However, it is also possible that the idiopathic form of acute delirium may be

produced by some unknown micro-organism that has a peculiar affinity to the central nervous system. This micro-organism then might not affect the other viscera of the body, but could produce extensive changes in the brain. SAILER.

42. ZUR LEHRE VON DER SPASTISCHEN UND INSBESONDERE VON DER SYPHILITISCHEN SPINALPARALYSE (Contribution to the Study of Spastic and especially of Syphilitic Spinal Paralysis). M. Friedmann (Deutsche Zeitschrift für Nervenheilkunde, 16, 1899, p. 140).

In the case described by Friedmann the symptoms began with thrombosis of the right retinal artery. A year later the gait became gradually weak and spastic, and after two and a half years only short distances could be traversed. The vesical functions and the cranial nerves were not affected. The patellar reflex was much exaggerated, foot clonus was present, and the paresis was greater in the left lower limb. The tactile sensation was more distinctly, though only moderately, impaired in the left lower limb. No marked subjective disturbances existed. The muscular power in the left upper limb was a little diminished. An apoplectic attack with left-sided paralysis occurred, and death followed five months later. An old focus was found in the right lenticular nucleus, and the basal arteries showed obliterating endarteritis. Degeneration was confined to the crossed pyramidal tracts, except in the lower thoracic region, where the direct cerebellar tract was affected, and in the cervical and upper thoracic region, where the anterior horn cells were moderately diseased on the side opposite to the cerebral focus. The degeneration of the pyramidal tracts extended from the lumbar region to the middle of the pons. The columns of Goll were not diseased. The involvement of the cells of the anterior horn on the side opposite to the cerebral focus was believed to be a part of the degeneration caused by this focus, and the case showed, by the normal condition of the cells in the right anterior horn, that anterior horn disease is not always associated with primary lateral sclerosis. Friedmann believes that all parts of the pyramidal tract throughout its degenerated portion underwent degeneration simultaneously before the hemiplegia occurred, and that a later involvement of the upper limbs was not a proof that the pathological process was ascending. The degeneration was believed by Friedmann to be syphilitic in origin, and the ground for this belief was the endarteritis. He acknowledges, however, that endarteritis may result from different causes, but in his case no cause could be found. SPILLER.

43. LES PHENOMENES DE REPARATION DANS LES CENTRES NERVEUX APRES LA SECTION DES NERFS PERIPHERIQUES (The Phenomena of Repair in the Central Nervous System After Section of the Peripheral Nerves). A. van Gehuchten (La Presse médicale, Jan. 4, 1899, p. 3).

In a previous communication, the author has reported his observations that after section of the peripheral spinal nerves, no chromatolysis occurs in the cells of the anterior cornua, but that it invariably occurs after the section of the cranial nerves. This has been criticised by Marinesco. Van Gehuchten reiterates his previous conclusion, and explains the method by which any error in observation was excluded. He believes that the experiments reported by Marinesco confirm in a decisive manner his own, although the interpretations put upon them by that author are very different. In regard to the second point, whether the continued separation of the nerve has an effect upon the rapidity of the reparatory processes, van Gehuchten expresses himself

as believing that it has not; neither his own investigations nor those of Marinesco having led to any very definite results. The union of the nerves, however, has a distinct effect upon the future of the ganglion cells. If it occurs, the cells not only become normal, but remain so; if, on the other hand, the nerve remains permanently separated, after apparently complete recovery, they subsequently undergo atrophy. Van Gehuchten has found, in opposition to Marinesco, that, during the first 15 or 20 days following the section, the cells gradually swell. After this there is a reformation of the bodies of Nissl. Van Gehuchten has also observed degeneration in the dorsal nucleus of the vagus after section of that nerve, and he has concluded that these cells are really motor in character.

SAILER.

#### THERAPY.

44. NOTE ON THE TREATMENT OF SEVERE CHOREA BY SULPHOCARBONATE OF SODA. T. Stacey Wilson (Birmingham Medical Review, 46, 1899, p. 1).

The paper contrasts the results of treatment in two cases of severe chorea. The first patient, 14 years old, was admitted to hospital one month after the beginning of the disease, having at the time constant movements of the face and all the extremities. She was treated first with chloralamide in doses up to 20 grains every three hours and then by chloral in large doses. Wet packs were also tried, but the patient rapidly failed and died of "asthenia" on the eighth day after admission. The temperature was subnormal throughout—96° to 97.5° F. The post-mortem examination revealed an endocarditis, and the erythematous flush on the cheeks and chest present during the latter days of the illness persisted after death.

The second patient was a young woman 22 years old, who had had two former attacks, the second one twelve months previously. She was admitted to hospital about six weeks after the inception of the disease, the case at the time not being very severe. She was put on Fowler's solution, with an occasional dose of chloral, but at the end of a week was much worse, the movements having become constant and decided mental symptoms having been added to the clinical picture. By the end of the tenth day in hospital she was completely helpless and almost unconscious. Believing that the condition was due to some infection, the author gave sodium sulphocarbolate and quinine as recommended by Carter of Liverpool and as found of value by the author in staphylococcus pyemia, viz.: twenty grains of the former, and one or two grains of the latter every two hours, alternately. The chloral was continued in occasional doses, as were also hot packs which had been used to promote perspiration. After thirty-six hours marked improvement began and recovery was rapid. The author mentions a "somewhat severe case" of chorea in a child treated with sulphocarbonates with a very satisfactory result, and a very severe case in an adult in which death occurred in spite of the same treatment.

PATRICK.

## Book Reviews.

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DIE EPILEPSIE. Professor Otto Binswanger. *Specielle Pathologie und Therapie*—Nothnagel. Vol. XII. Alfred Hölder, Wien, 1899.

Binswanger defines epilepsy as a definite chronic disease of the central nervous system, produced by the most varied causes. Its pathological effects consist in either frequently recurring convulsive attacks with unconsciousness, or in modifications of such attacks, or in psychopathic symptoms accompanying or resulting from them.

In the chapter on general pathology and pathogenesis, the theories of Brown-Séquard, Hitzig, Bubnoff, Heidenheim, Jackson and Gowers; and the author's own experiments on dogs, and their applications to human beings, are considered. The author comes to the conclusion that in genuine epilepsy the origin of the irritation, which gives rise to the epileptic attacks, is to be sought in a primary irritation of the cortex, but that the result of this stimulation, in respect to the discharge of the convulsive components, reaches most quickly and most intensely its effect in the infracortical motor central apparatus. The assumption of an original cortical irritation is based upon the mental disturbance of consciousness and the majority of the forms of aura. The following six conclusions are noted as the result of a consideration of the pathological and physiological data stated in the chapter devoted to this subject: First, the seat of the epileptic cortical changes must be thought of as involving the brain as a whole, although the disturbed activity of the cortex, in consideration of its high functional qualities, occupies the most important position in the process. Second, the nature of the epileptic changes is yet unknown. Third, the epileptic discharge causes in the main two pathological conditions, which may be best characterized as inhibitory and excitent discharges. Fourth, the typical epileptic attack is one of the expressions of the disease. This originates most probably from a primary cortical discharge, but a concomitant stimulation of the infracortical motor central apparatus (basal ganglia, corpora quadrigemina, pons and medulla) is essential for the full development of the attack. Fifth, in the incomplete and petit mal form, the discharges can be limited to the cortex alone, or be confined to one of the infracortical central apparatus. Sixth, the convulsions of cortical origin, as concomitant symptoms of organic diffuse or focal disease of the brain have only an indirect relation to epilepsy.

The author discusses the etiology under two heads: first, the cause of the disease as a whole; second, the cause of the individual convulsions. The greatest weight under the first head is laid upon what Griesinger calls neuro- or psychopathic predisposition, which may be defined as a state of lessened resistance to pathological and physiological irritation. This condition may be directly hereditary, or intra-uterine acquired or extra-uterine acquired tendency. The inherited disposition may be due to some disturbing germinal cause on the part of the parents, as chronic intoxication, alcohol, morphine, lead, etc.; infections, such as syphilis, tuberculosis, and constitutional diseases; and lastly local diseased conditions of the germ-producing organism.

The main cause of the intra-uterine acquired disposition is trauma of various kinds affecting the fetus. The conclusions arrived at by studying large numbers of statistics is that 35 to 40 per cent. of epilep-

tics have an hereditary etiology. If the other causes mentioned above are considered, this percentage would be considerably increased. The relation between infantile eclampsia and the later developing epileptic attacks is thoughtfully considered by the author, and its importance is brought out very forcibly. The author believes that infantile convulsions in the first few weeks of the child's life, are indications, as a rule, of the child's lessened resistance. In this sense the eclampsia is not only a forerunner, but an actual cause of epilepsy. The important question of inherited syphilis and epilepsy is given a prominent place and numerous cases are cited showing the actual etiological relation. Trauma is a very important causative factor. The cause of the first attack, from the cause of the subsequent ones, should be sharply differentiated. If an epileptic attack is once caused by any of the previously mentioned factors, then the attacks may be repeated indefinitely, independent of a repetition of the first cause. It is always true, however, that the first cause produces a stronger effect upon the brain than any of the later ones. Psychical effects of one kind and another, such as fright, emotional excitement, etc., are found to exist as the first cause in about 75 per cent. of the cases.

Symptomatology.—The following divisions in regard to the symptoms are noted: first, the fully developed attack (*epilepsia gravior*, *haut mal*); second, rudimentary epileptic attack; third, abortive attack (*epilepsia mitior*, *petit mal*); fourth, the psychical epileptical equivalent (*epilepsia larvata*, or psychical epilepsy). The classical attack has three stages: prodromal, convulsive, and the soporific after-stage. Under the prodromal symptoms the aura is given the most important place. The author divides the aura into psychical, sensory, sensible, motor and vaso-motor. The condition of the pupils during the attack is no longer pathognomonic; the absence of the light reflex was formerly thought to be so typical of an epileptic attack that it could, by this symptom alone, be distinguished from hysterical convulsions. This is no longer true, as some epileptics retain the light-reflex, and in hysterical attacks often an absence of reflex occurs. Abortive attacks, *petit mal*, are defined as attacks in which the motor convulsive component is wanting or merely indicated, while the disturbance of consciousness attracts the attention of the observer; or attacks in which disturbance of consciousness is absent and only brief motor excitement or explosions of inhibition form the condition. Under the first division, the epileptic vertigo, or *absence* of the French, is included. Two divisions of this type of epilepsy are noted: 1. *Petit mal* intellectual, and 2, *grand mal* intellectual. Cases of retrograde amnesia are found in epilepsy. Post-epileptic stupor (*Dämmerzustand*) form the forensic importance of this question.

The pathology of epilepsy, always the weak point in works on this disease, finds no exception here. Outside of the findings of various cerebral defects in structure and other gross changes, nothing very definite concerning the microscopical examination has as yet been discovered. Even with the aid of Nissl stain no constant cell changes have been found. Concerning the much debated question of glia hypertrophy in epileptic brains, the author holds that its importance has been much exaggerated. He was unable to demonstrate this condition in his own examination of epileptic brains. The theory of Chaslin, that essential epilepsy is caused by the proliferation of the neuroglia, even if no definite lesion is present, is regarded by the author as by no means proven; firstly, on the grounds of too small a material; secondly, on the possibility that the diagnosis of idiopathic epilepsy may not be correct. Alzheimer's theory, that the neuroglia is patho-



logically changed in structure; Bevan Lewis's theory, that the ganglion cells are changed in structure; and many other numerous theories are all regarded as insufficiently proven. More important at present is the study of the irregularity, in the form of the skull and brain, as was first done in great detail by Benedict of Vienna. Under differential diagnosis, the usual means of differentiating hysteria and epilepsy are noted. The weight that was formerly placed upon the pupillary reaction is not insisted upon by the author. More attention is laid upon the psychical changes found so frequently in epilepsy and so seldom in hysteria, if the disease has lasted a long time. A. Voisin regards the pulse and respiration as two valuable differential signs. It is to be remembered that not seldom a true epileptic may simulate attacks; this is a point that has been too little regarded.

In regard to therapy, there is contained such a wealth of material that it is very difficult to make an abstract of it. The best prognosis is offered in cases of children between the ages of six and fourteen, where no organic lesion lies at the bottom of the disease; also in cases between fifteen and thirty years, when the attacks have developed after puberty and no mental defect has, as yet, resulted. As most hopeless are those cases which have shown, previous to the onset of epilepsy, evidences of mental enfeeblement, or where this condition begins with or shortly after the first attack. The therapy is divided into three divisions: Constitutional, that is hygienic and dietetic, medicinal, and operative. The first importance is given to the bromides, the successful use of which depends upon the long period of administration. The dose must be regulated according to the individual and to the nature of his epilepsy. The brom-opium treatment of Flechsig is to be recommended only to patients in institutions, or those who are under the direct care of a skilled physician or nurse. No definite opinion is expressed concerning the value of this method, as the author believes that the time has been too short. The operative interference is treated judiciously and in the light of the newer ideas on the subject. Binswanger believes that only purely reflex types of epilepsy are suitable for operative intervention.

In every respect this work is a valuable contribution to the literature of epilepsy, superior in many ways to the work of Féré and Voisin, chiefly in the more generous and the wider acknowledgment of the work of foreign investigators. One is struck in reading the work by the great personal experience and the fair judgment and the absolute lack of prejudice of the author. The grouping together of facts and theories from every authority is truly marvelous. The careful and precise definitions of the various phases of the disease is a valuable feature of the work. It is a compendium of all the knowledge that we have on epilepsy, beautifully arranged and clearly stated, and carrying with it that priceless virtue of all true scientific work, the touch of personal experience. The work is most earnestly recommended to those who have an interest in the question of epilepsy and its manifold aspects, and who desire a broad knowledge of the subject. A good bibliography and a fair index make the book valuable for reference.

SCHWAB.

## MISCELLANY.

### AMERICAN NEUROLOGICAL ASSOCIATION PRELIMINARY PROGRAM.

#### OF PAPERS TO BE READ AT THE TWENTY-SIXTH ANNUAL MEETING

TO BE HELD IN

WASHINGTON, D. C., MAY 1, 2 and 3, 1900.

On the morning of May 2d there will be a discussion on the "Neuron Doctrine in Its Relationship to Diseases of the Nervous System." The subject will be subdivided as follows:

1. "The Anatomico-cytological Relationship of the Neuron to Disease of the Nervous System," By Dr. L. F. Barker, of Baltimore.
2. "The Pathological Changes in the Neuron in Nervous Disease." By Dr. William G. Spiller, of Philadelphia.
3. "How Far Does it Affect Our Conception of Nervous Disease and Its Treatment." By Dr. B. Sachs, of New York.
- "Autopsy of a Case of Adiposis Dolorosa, with Microscopical Examination." By Dr. F. X. Dercum, of Philadelphia.
- "Studies in Astereognosis." By Dr. F. X. Dercum, of Philadelphia.
- "A Case of Wernicke's Conduction Aphasia, with Autopsy." By Dr. Howell T. Pershing, of Denver.
- "Amelioration of Paralysis Agitans by Means of Systematized Exercises." By Dr. J. Madison Taylor, of Philadelphia.
- "Christian Pseudo-science and Psychiatry." By Dr. Smith Baker, of Utica.
- "Epilepsy and Auto-intoxication." By Dr. Smith Baker, of Utica.
- "Cases of Neurasthenia." By Dr. Philip Zenner, of Cincinnati.
- "Syringomyelia with Exhibition of Microscopical Specimens." By Dr. Greame M. Hammond, of New York.
- "A Case of Polio-encephalitis, with Autopsy." By Dr. Chas. L. Dana, of New York.
- "Tumor of the Superior Parietal Lobule, Accurately Localized and Removed by Operation." By Dr. Chas. K. Mills and Dr. W. W. Keen, of Philadelphia.
- "Erythromelalgia and Allied Disorders." By Dr. B. Sachs, of New York.
- "Some Unusual Types of Dementia Paralytica." By Dr. Theodore Diller, of Pittsburgh.
- "Contribution to the Study of the Plantar Reflex. Based Upon Seven Hundred and Fifty Tests, Made with Special Reference to the Babinski Phenomenon." By Dr. G. L. Walton, of Boston.
- "A Case of Malaria, Presenting the Symptoms of Multiple Sclerosis, with Necropsy." By Dr. Wm. G. Spiller, of Philadelphia.
- "A Case of Adiposis Dolorosa, with Autopsy." By Dr. Charles W. Burr, of Philadelphia.
- "A Case of Multiple Sclerosis, with Autopsy." By Dr. Charles W. Burr and Dr. D. J. McCarthy, of Philadelphia.
- "Two Cases of Tumor of the Spinal Cord." By Dr. John Jenks Thomas, of Boston.
- "Demonstration of a Few Reconstructions of Parts of the Nervous System." By Dr. Adolf Meyer, of Worcester.

- "Communication on a Comparative Study of the Spinal Ganglia in Various Diseases and Ages." By Dr. Adolf Meyer, of Worcester.
- "Clinical and Anatomical Analysis of Cases of Diffuse Myelitis." By Dr. James J. Putnam and Dr. E. W. Taylor, of Boston.
- "Report of a Case of Progressive Muscular Atrophy, which Clinically Presented the Symptoms of Amyotrophic Sclerosis." By Dr. Joseph Collins, of New York.
- "A Clinical Study of the Reflexes." By Dr. Joseph Collins and Dr. Joseph Fraenkel, of New York.
- "The Physiologic and Therapeutic Effect of Sunlight." By Dr. Frank Hallock, of Cromwell.
- "Report of a Case of Brain Injury with Peculiar Whistling Spells Following Operation." By Dr. William C. Krauss, of Buffalo.
- "Imperative Ideas in the Sane and Their Management." By Dr. Edward B. Angell, of Rochester.

PRELIMINARY PROGRAM OF THE SECTION ON NEUROLOGY AND MEDICAL JURISPRUDENCE OF THE AMERICAN MEDICAL ASSOCIATION TO BE HELD AT ATLANTIC CITY, JUNE 5-8, 1900.

1. Address of the Chairman, Hugh T. Patrick, Chicago.
2. "A Report of Seven Operations for Brain Tumors and Cysts." Herman H. Hoppe, Cincinnati.
3. "Treatment of Neurasthenia." D. R. Brower, Chicago.
4. "The Diagnosis of Apoplexy Without Motor Paralysis," Wm. N. Bullard, Boston.
5. "On Certain Routine Treatment for Gouty Conditions," Frank R. Fry.
6. "Legal Responsibility of Degenerates Not Insane," David Inglis, Detroit.
7. "Travel Therapeutics in Nervous and Mental Maladies," Richard Dewey, Chicago.
8. "The Differentiation of Chorea and the Disorders Simulating It," A. A. Eshner, Philadelphia.
9. "Post-febrile Insanities and Their Treatment," Frank P. Norbury, Jacksonville.
10. "The Modern Treatment of Locomotor Ataxia," Curran Pope.
11. "A Case Resembling One of Raynaud's Disease, with Microscopic Examination," Wm. G. Spiller, Philadelphia.
12. "Certain Means for Relief of Neuralgia," W. J. Herdman, Ann Arbor.
13. "Combined Sclerosis of Putnam-Dana Type," F. W. Langdon, Cincinnati.

14. "Nature and Symptomatology of the Traumatic Neuroses," Harold N. Moyer, Chicago.
15. "The Nervous Diseases Caused by Trauma," Chas. W. Burr, Philadelphia.
16. "Traumatic Neuroses from the Standpoint of a Railway Surgeon," Arthur Dean Bevan, Chicago.
17. "Medico-legal Relations of Traumatic Nervous Affections," Landon Carter Gray, New York.
18. "Prognosis and Treatment of the Traumatic Neuroses," Wharton Sinkler, Philadelphia.
19. "An Analysis of Cases of Traumatic Neuroses with Special Reference to Prognosis," Jas. J. Putnam, Boston.

Discussion to be opened by Jas. Hendrie Lloyd, H. H. Hoppe, and F. X. Dercum.

20. "Diagnosis of Hysteria from Organic Disease of the Brain," Chas. K. Mills, Philadelphia.
21. "Diagnosis of Hysteria from Organic Disease of the Spinal Cord and Peripheral Nerves," Frederick Peterson, New York.
22. "General Treatment of Hysteria," B. Sachs, New York.
23. "Technique of the 'Rest-Cure'—Indications and Contra-indications," John K. Mitchell, Philadelphia.

Discussion to be opened by Chas. W. Burr and William G. Spiller.

24. "Medico Legal Relations of Opium Inebriates and Necessity for State and Interstate Statutes," T. D. Crothers, Hartford.
25. "Aphasia, with Report of a Case," Guy Hinsdale, Philadelphia.
26. "True Epilepsy with Symptoms of General Paresis Engrafted: Report and Presentation of a Case," A. Ferree Witmer, Philadelphia.
27. "Cranial Injuries and Insanity, with Report of a Case," E. G. Carpenter, Columbus.
28. "Prophylaxis of Chorea," J. Madison Taylor, Philadelphia.
29. "Migraine—Report of a Case and Consideration of Heredity," D. J. McCarthy, Philadelphia.
30. "Involutional and Evolutional Types of Nervous Disease," Ed. E. Mayer, Pittsburg.
31. "Presentation of Some Specimens of Brain Tumor," A. E. Sterne, Indianapolis.
32. "Morphinism from the Standpoint of the General Practitioner," T. J. Happel, Trenton, Tenn.
33. "Hereditary Color Blindness," F. Savary Pearce, Philadelphia.
34. "The Significance of Certain Factors in Family and Personal Histories, and Their Relation to Nervous Disease," John Punton, Kansas City.
35. "Report of a Case of Brain Tumor," Chas S. Potts, Philadelphia.
36. "Post-anesthetic Paralysis," C. C. Hersman, Pittsburg.

F. SAVARY PEARCE, Secretary, 1407 Locust street, Philadelphia.

#### BOOKS RECEIVED.

"Nervous and Mental Diseases," by Chas. S. Potts, M.D. Edited by Bern. B. Gallaudet, M.D. Lea Bros. & Co., Phila. Pa.

"The Treatment of Diseases of the Nervous System," by Joseph Collins, M.D. Wm. Wood & Co., New York.

"The American Year-Book of Medicine & Surgery," by Geo. M. Gould, M.D., two volumes, Medicine & Surgery. W. B. Saunders, Phila., Pa.

"A Text-Book of Diseases of Women," by Chas. B. Penrose, M.D. W. B. Saunders, Phila., Pa.

"A Manual of the Diagnosis and Treatment of the Diseases of the Eye," by Edward Jackson, M.D. W. B. Saunders, Phila., Pa.

"Letter-, Word- and Mind-Blindness," by James Hinshelwood, M.D. H. K. Lewis, London, Eng.

"A Manual of the Practice of Medicine," by A. A. Stevens, M.D. W. B. Saunders, Phila., Pa.

"Christian Science—An Exposition," by Wm. A. Purring-ton, M.D.

"Baking Powders," by Chas. A. Catlin, M.D. Rumford Chem. Co., Providence, R. I.

"Proceedings of the American Medico-Psychological Association at the Fifty-fourth Annual Meeting held in St. Louis, May 10-13, 1898."

"Transactions of the Iowa State Medical Society," Vol. xvii. Forty-Eighth Annual Session, 1899.

"St. Bartholomew's Hospital Reports." Vol. xxxv. Smith Elder & Co., London, Eng.

"Anatomie clinique des centres nerveux," par Le Dr. Grasset. J. D. Bailliere et Fils, Paris, France.

Separat-Abdruck aus "Handbuch der praktischen Medicin." Dr. W. Ebstein and Dr. J. Schwalbe.

"Die Literatur der Psychiatrie, Neurologie und Psychologie von 1459-1799." In three volumes. Herausgegeben von Dr. Heinrich Laehr. Berlin, George Reimer, 1900.

"Mentally-Deficient Children. Their Treatment and Training." By G. E. Shuttleworth, B.A., M.D. Second Edition. P. Blakiston's Son & Co., 1900.

THE  
**Journal**  
OF  
**Nervous and Mental Disease.**

**Original Articles.**

INAUGURAL ADDRESS BEFORE THE AMERICAN  
NEUROLOGICAL ASSOCIATION.

BY THE PRESIDENT, EDWARD D. FISHER, M.D.

Gentlemen: Before bringing to your attention the principal subject of my remarks, as I open another annual meeting of our association, I would take the opportunity to express my appreciation of the honor conferred on me by my election as President of the American Neurological Association, and especially at this time, when the Congress of American Physicians and Surgeons holds its triennial at Washington.

The position of neurology among the divisions of medicine and surgery has changed very much in the last twenty or twenty-five years. Perhaps no special branch of medicine comes into quite so close touch with general medicine and medical surgery as neurology. The work which has been done by many in our own field in this country, those living and dead, has also established neurology on as high a level of honor and respect as any of the departments of medicine. This was not always so, as in our special field there has ever been an opportunity for the charlatan to exercise his special talents.

The great progress in our knowledge of the anatomy and pathology of the nervous system, the acquirement of which knowledge calls upon the highest kind of intellectual study, has had much to do with this.

In the old world, owing largely to the clinics held at the

medical colleges, the reputation of the various teachers became world-wide. It is, however, only within the past quarter of a century, if so long, that the medical colleges with us have recognized the paramount necessity of a full and thorough course in psychiatry and neurology. This implies at present that in every well-organized college a previous thorough training in the minute anatomy of the nervous system has become necessary. Without this latter no proper instruction can be given in disease of the nervous system.

A very important point for us to consider, if we wish this high standing in the community to be maintained, is that we must not separate ourselves from general medicine. We must also study general disease. Other specialties may remain narrow and limited, but it should not be so with ours.

We are more often than others called as a sort of last resort to cases after many opinions have been expressed. Our diagnosis must be one often by exclusion. We must, therefore, be able to recognize, and, indeed, must fully understand, general diseases. We should be as able to examine the heart and lungs and other internal organs, and to recognize disease in them, as we are to make out disease of any special character in the nervous system itself. Again, a reason which also applies here is that these diseases of the general system may influence or cause the affections of the nervous system. A special illustration of this is seen in syphilis, which has such well-defined effects upon the nervous system. Some of the greatest names in neurology in Europe have always been connected with general medicine. I do not think it advisable to continue, in general practice at least, to take care of any of the acute diseases, although some of the chronic diseases may often come under our care.

Another subject of importance for us to consider is that of therapeutics. We are too often, perhaps justly, accused of being more interested in the diagnosis and localization of a disease, and in the findings at autopsy, than in its cure. In calling us as consultants, however, the physician and patient are looking not only for a diagnosis from us, but also for some suggestion in regard to treatment. One of our most celebrated neurologists, Dr. S. W. Mitchell, by his practical carrying out of a

scheme or plan of treatment for nervous disorders which will always connect his name with the rest cure, has done much for the treatment of disease and also has spread the name of American neurology the world over.

I do not underrate the importance and necessity of the continued study of the histology and pathology of the nervous system. Our real advance is mostly due to this study and all our treatment has depended on such knowledge.

The note of warning I would now sound, and especially to those about to enter this field of medicine, is, keep up as broad a knowledge of all medicine as possible. No one should enter neurology as a specialty without having first had an experience in general practice.

I believe if I read the signs of the times aright that is the tendency of the neurology of the future.

Now we will enter upon the work of the session. We have an unusually full and excellent number of papers. Our association has been successful and fortunate in its additions to its membership. We have now some most excellent candidates awaiting admission. It will be necessary to increase our membership, and from the attainments of those now applying I think it would be doing the association a distinct injury not to so enlarge it. To continue to do good work we must ever be taking new blood into our association.

Again thanking you for the high honor conferred upon me I declare this session now open for scientific work.

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45. OEDEME DYSTROPHIQUE (Dystrophic Edema). A. Vigouroux (Nouvelle Icon. de la Salpêtrière, Nov., Dec., 1899).

A description of a case of dystrophic edema of the left leg. The whole leg from the thigh to the toes was affected by a white, hard and painless edema. The difference in circumference between the two legs was very marked. The skin was white without excoriations, and glistening, and varicosities were present. The skin did not pit on pressure; was colder to the touch than the normal leg. The skin increased in hardness during the last five or six years. Sensibility was intact. The motility of the leg was well preserved. Patient's gait perfectly normal, and no unusual fatigue resulted from walking. Other organs were normal. The condition developed at the age of puberty. No hereditary history was obtainable. The article is illustrated by photographs of the case. SCHWAB.

TUMOR OF THE SUPERIOR PARIETAL CONVOLUTION,  
ACCURATELY LOCALIZED AND REMOVED BY  
OPERATION.\*

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AND

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PROFESSOR OF THE PRINCIPLES OF SURGERY AND OF CLINICAL SURGERY,  
JEFFERSON MEDICAL COLLEGE.

WITH A PATHOLOGICAL REPORT ON THE NATURE OF  
THE GROWTH.

BY W. G. SPILLER, M.D.,

PROFESSOR OF DISEASES OF THE NERVOUS SYSTEM IN THE  
PHILADELPHIA POLYCLINIC.

MEDICAL HISTORY BY DR. MILLS.

It will probably be admitted that the following case is one of unusual interest from the clinical, physiological and surgical points of view. The diagnosis of the case naturally gave rise to differences of opinion, chiefly because of the absence of some of the most frequently observed manifestations of organic focal disease and the presence of other symptoms and signs commonly regarded as characteristic of functional nervous disorder. The difficulties of diagnosis between grave hysteria and serious organic focal disease of the brain were in the foreground at several stages in the history of the case. Physiologically the case is important because it has added something to our knowledge of the areas of representation of muscular and cutaneous sensibility and of trophic functions. Dr. W. W. Keen will consider the surgical aspects of the case, and I shall only say here that the operation in method and execution was one of the most brilliant of the many that it has been my good fortune to see. The following is the history of the case in detail:

Mr. S. R., fifty-seven years old, enjoyed good health until 1884, when he became very nervous, emotional and despondent, and began to suffer from headaches chiefly in the back of the head. One physician whom he consulted at this period

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\*Read at the twenty-sixth annual meeting of the American Neurological Association, May, 1900.



told him that he had beginning softening of the brain, a diagnosis which exercised a depressing influence upon him. He went to Europe, spending a few weeks at Kissingen and about twenty-two months at Frankfort-on-the-Main, where he lived an easy and quiet life and recovered almost entirely, except that he had an occasional headache. He returned to America and to his business in 1886, and for eight years from that time he remained in comparatively good health.

In 1894, while walking on the street, he suddenly felt an unpleasant sensation in his right arm, which he described as a feeling as if a mouse or ants were creeping down his arm. After this, sometimes at intervals of days, and at others of weeks, or even months, he had similar paresthetic attacks in the right arm; these were sometimes described as a tingling or battery sensation. They came on suddenly and passed away in a few moments. After the attacks the arm would feel somewhat heavy. During the attacks he was never unconscious and so far as could be learned he never suffered from vertigo. From this time on he was more or less neurasthenic and hysterical, with periods of great despondency. He suffered much with a feeling of pressure or distress in the head, which he usually referred to the left parieto-frontal region. At times it took on the character of a true localized headache, but this was exceptional, the uncomfortable sensation usually being described by the patient as a feeling of pressure or distress. He was fully able to attend to his business until January, 1899, although he was often nervous and did not feel well. In 1894 he consulted Professor Eichhorst; he was also seen by others, but so far as I could learn the diagnosis of organic brain disease was not made, except the opinion expressed in 1884 that he had softening of the brain, which in the light of his subsequent history was evidently without good foundation.

In June, 1899, he first noticed that he was beginning to use his right upper extremity awkwardly, and a week or two later a similar awkwardness of movement became noticeable in his right leg. From this time he had slowly increasing difficulty in the use of his right arm and right leg, and especially the former. The disability became markedly worse about the middle of August, and was described by the patient as a loss of power, although my first examination of him, made August 21, showed that the disorder in the limbs of the right side was ataxic rather than paretic. He had no pain in the affected extremities; but he complained frequently of a feeling of pressure in the left parieto-frontal region. He also had frequent attacks of a hysterical character with great despondency.

My first examination of the patient on August 21, 1899,

showed that he had distinct but not very marked ataxia both in the right upper and lower extremities, the ataxia being more marked in the arm. Sensation to pain, touch and temperature was impaired over the right half of the body, and he had distinct astereognosis in the right hand. Muscular power was good in the right arm and leg, although the patient complained of a feeling of heaviness in these parts.

Between August 21 and September 25 I saw the patient three or four times. The ataxia of the extremities got gradually worse, and he daily complained of a sensation of pressure or pain, which he always referred to the left parieto-frontal region. On questioning him closely about the sensation he would usually state that it was not exactly pain but rather a feeling of pressure or distress, although at times he referred to it as a pain or even as a headache. From September 25 I saw the patient almost daily, and not infrequently twice or oftener in a day. About this time I first expressed the opinion that he was probably suffering from a slowly developing brain tumor.

Frequent examinations were made after he came under constant observation, the results being similar to those obtained September 29, when he was seen in consultation with me by Dr. F. X. Dercum and Dr. Wm. G. Spiller. The following points were recorded at this time:

Hypesthesia to touch, pain and temperature was present on the right side of the body; face, trunk, arm and leg all being examined. Sensation was nowhere completely lost, although distinctly impaired as determined by numerous tests. Sensation was a little less impaired in the right side of the nose and in the right cheek near the nose than in other regions in this half of the body. The mucous membranes on the right, as well as the skin, showed partial anesthesia. The right conjunctival reflex was diminished. Examination for hearing, smell and taste showed also some slight diminution, a result which was subsequently not uniformly obtained. Frequent tests were made with his right hand as to the condition of the stereognostic sense. He was found to have distinct astereognosis in the right hand. Ataxia of both the upper and lower extremity on the right side was very marked, and he was also somewhat paretic in both, but more so in the leg than in the arm. The knee-jerk, quadriceps-jerk, and other tendon and muscle phenomena on the right side were slightly exaggerated, and he had distinct but soon disappearing ankle-clonus on the right side; toe-jerk and front-tap were absent. The Babinski reflex at this time was also absent, irritation of the ball of the foot causing plantar flexion. The patient at the time of the exami-

nation and almost continuously at and after this period showed many hysterical symptoms.

Almost daily examinations were made subsequent to this time. The patient's condition grew slowly worse. The notes made from October 5 to 9 showed about the following conditions: He continued to have diminished sensibility to pain, touch and temperature in the right half of the body, not notably worse than when previously examined. The tests for hearing, smell and taste showed nothing of a decisive character, although taste at times seemed blunted on the right side and at others on both. The right half of his tongue was continuously more coated than the left. The ataxia of arm and leg became more and more marked, and the loss of power in these limbs was slowly deepening into a profound paralysis. Astereognosis was now of the most decided character. While the knee-jerks were not markedly different on the two sides, persistent ankle-clonus was now always obtainable on the right. The Babinski reflex could sometimes be obtained on the right and sometimes not, but even when the plantar response was normal the flexion of the toes was not as active on the right as on the left. The toes of the right foot were now continuously in a state of partial metatarso-phalangeal dorsal flexion.

It was noted about October 9 that for a time that could not be accurately fixed the patient had shown some amnesia for names and words. He frequently could not recall the names of his recent or earlier physicians, and occasionally he was unable to recall the names of familiar objects. Because of this difficulty he was at times unable to continue a consecutive conversation. His amnesia varied greatly, however, at different times, and seemed to be considerably affected by his emotional state.

On October 9 he was tested as to his ability to read, using a few lines from a newspaper and a poem containing chiefly words of one or two syllables. He read slowly and with effort, but did not mispronounce the words. He was quickly fatigued by the reading. Now and then he complained of some disturbance of vision, but this was not persistent and may have been due to some defect of refraction.

He continued to complain of a feeling of pressure or pain, or of something wrong in his head, usually putting his hand at these times to the left side of his head near the vertex; occasionally he complained of occipital pain, but his statements regarding pain in the head were vacillating.

On several occasions in October he was examined by Dr. S. D. Risley, who reported some disorder of refraction, but that optic neuritis was not present. The tests for his color fields

were somewhat confusing, at times showing reversals and at others not. The fields for form and color were however contracted. At one examination he had, or appeared to have, a partial right lateral homonymous hemianopsia, but when examined again this could not be determined. Wernicke's symptom was not present.

Some paresis of the right side of the face and a slight deflection of the tongue to the right were now observable. The soft palate was also deflected a little to the right. During October and November the patient's pulse always ranged below normal; on several occasions it fell as low as forty to the minute, and most frequently varied between 45 and 60. Inquiry elicited the fact that the patient's pulse was slow when he was in good health, probably ranging in the neighborhood of 60. As a rule the patient presented no pupillary or other ocular symptoms, but on October 17 an inequality of the pupils was observed, the right being larger than the left. This inequality remained only a day or two. Although the patient's symptoms were chiefly right-sided, some interesting observations were made from time to time with regard to the conditions present in the left half of the body. The left leg remained practically normal; the movements of the left arm however showed some incoordination, as determined by the usual methods of having the patient with his eyes closed touch various parts of the face and pick up objects of different size. The ataxia was comparatively slight and seemed to be aggravated by the patient's emotional and hysterical condition. No objective cutaneous or muscular anesthesia and no astereognosis were ever detected in the left half of the body, but late in October the patient complained of a feeling of numbness in the left hand, a symptom which remained until after the operation. He continued to be seen at intervals by Drs. Dercum and Spiller in consultation. For several weeks late in October, apparently as the result of the use of mercurial inunctions and the iodides, he made some improvement in the use of his arm and leg, but about November 1 he had lost all he had gained and in a few days was distinctly worse. The paralysis especially became more and more profound and his use of language more restricted. Some wasting was noticeable, especially in the right upper extremity. On November 18 he had what appeared to be a slight apoplecticiform attack in which he became much confused and agitated, and lost his speech entirely for about an hour, but he was not unconscious.

He was seen in consultation November 19 by Dr. James Hendrie Lloyd and Dr. William Osler. Paralysis of the right arm and leg was now almost absolute. The face showed only

slight paresis. The ataxia of the limbs of the right side was now masked by the completeness of the paralysis. Hypesthesia as to touch, pain and temperature was about the same as on numerous previous examinations. Right ankle-clonus and the Babinski reflex were present, otherwise the deep reflexes were not much altered. The muscles of the hand, forearm, arm, shoulder, breast and leg of the right side showed distinct atrophy. He had extreme difficulty in recalling words; he could not keep up a consecutive conversation and only with difficulty answered questions regarding himself. He complained, as often before, of pain on the top and left side of the head. The sphincters of the bladder and bowels showed some tendency to relaxation, and he was in a distressing emotional state.

November 20 the patient was taken to the private hospital of Dr. W. W. Keen, operation for the removal of a brain tumor having been decided upon. The operation was fixed for November 24, and the day previous a final consultation regarding the case was held, those present and taking part being Drs. W. W. Keen, F. X. Dercum, J. H. Lloyd, W. G. Spiller, S. D. Risley, J. W. McConnell and the writer. A re-examination of the patient was made at this time, but this developed no new symptoms or conditions. The paralysis, anesthetics, speech defect, reflexes, and emotional state remained as given in the immediately preceding notes. Dr. S. D. Risley made a final critical examination with the ophthalmoscope and reported the absence of all evidences of optic neuritis.

Two months previous to the date fixed for the operation I had diagnosticated brain tumor, and then and subsequently had fixed upon the left superior parietal lobule as its probable site, although I regarded the growth as most likely originating in the subcortex. With the accord of my consulting colleagues it was decided to operate with the view of first uncovering this portion of the brain. The localization was in the first instance based upon loss of the muscular sense, impaired cutaneous sensibility, astereognosis and ataxia. It may be well here to briefly summarize the data which led both to the general diagnosis and to the localization of the growth; later the most important points in the case will be discussed in more detail.

About five months previous to the operation the patient began to show some ataxia in the right arm and later in the

right leg, and when investigation of his condition was first made by the writer all forms of cutaneous sensibility were impaired, muscular sense was lost, and astereognosis was a marked symptom. As the case progressed paresis and eventually paralysis of the arm and leg supervened, this when complete of course masking the ataxia. The patient developed a disorder of speech chiefly showing itself as a verbal amnesia and fatigue on reading. At one examination the patient showed a temporary partial right hemianopsia. Reversals of the color fields and contractions of the fields for form similar to those supposed to be typical of hysteria were present at several of the examinations. The reflexes on the ataxic and paralyzed side were somewhat exaggerated, ankle-clonus being present. The patient was emotional and markedly hysterical. The general symptoms of brain tumor were not only not prominent but the most important of them were absent. The patient had not the typical severe headache so often present in cerebral neoplasms, although he complained much of feelings of discomfort, distress and pressure, and occasionally of pain, these sensations being almost uniformly referred to the left parietal or parieto-frontal region near or about the median line of the head. Vertigo, nausea and vomiting were not symptoms, and optic neuritis was entirely absent as shown by repeated careful examinations. The patient from first to last had no convulsions, and not even the slightest local spasm. In spite of the difficulties in the way of correct diagnosis it was believed that the case was one of brain tumor originating in what the writer holds to be the true cerebral sensory area, this opinion being based chiefly on the sensorial localizing symptoms and on the pressure symptoms which ensued as the growth enlarged in size and the case developed. The visual symptoms, the disorder of language, the motor paralysis, and the changed reflexes were thought to be in the main pressure symptoms, although it was believed that the motor subcortex had probably been invaded to some extent.

An operation which was successfully performed by Dr. W. W. Keen November 24 exposed a tumor in the exact region which had been assigned as the seat of the growth. The details of the operation, which was in many respects of unusual

interest, and the subsequent surgical progress of the case, will be described by Dr. Keen.

The patient was fully conscious about three hours after the completion of the operation, although weak and nauseated. Dr. Keim, who was constantly with the patient as nurse and attendant for several weeks before and after the operation, recorded that, after coming out of the ether, the patient could talk freely. Within forty-eight hours he could not speak more than a very bad (indistinct) "yes" and "no"; in seventy-two hours he could say "yes" and "no" plainly; at ninety-six hours he could make sentences of three or four words, as "I want my wife." Five days later he could make sentences of six to ten words.

On the day after the operation he was examined by me, but only briefly, I believing it best to avoid any prolonged investigation of his condition. He was weak and complained of pain in the head in the region of the operation, and also of some pain shooting down the right side of the neck and down the arm of the paralyzed side. Both arm and leg were completely paralyzed, but there was no paralysis of the face. Sensation to touch and pain were diminished on the right side more markedly than before the operation. He remained much the same for several days, his general condition, however, slowly improving. On November 28 temperature and pulse were about normal, his respiration good, and the case in general was progressing favorably. On the 29th he talked more freely than at any time since the operation.

On December 2, eight days after the operation, slight motion was noticed for the first time in the right leg. Each day this ability to move the leg slightly increased, and by December 4 he was able to partially flex the thigh on the pelvis and the leg on the thigh. During the week from December 4 to December 11 he daily gained additional power in his right leg. As just stated, he was first able to flex the thigh on the pelvis and the leg on the thigh; next to thrust or push downward the leg, and later to carry the right leg toward and finally over the left. About the 7th day after the first return of power he was able to slightly flex the toes; at first he could dorsally flex the great toe; dorsal and plantar flexion of the toes came a few days later. Sensibility to touch, pain and temperature continued to be greatly impaired on the affected side, but it was nowhere absolutely abolished. Moderate right ankle-clonus was present; the knee-jerk was slightly exaggerated and the plantar reflex seemed to halt midway between normal plantar flexion and the Babinski reaction. His speech was improving, but vacillated considerably, and was without doubt greatly in-

fluenced by his emotional state. December 6 a slight return of power was first observed in the upper extremity. The first return observed was a slight pushing or extension movement of the entire arm backward and forward. He had frequent attacks of depression and emotional excitement due to apprehension.

On December 11 I made a careful examination of the patient in conjunction with Dr. Wm. G. Spiller. The patient sat up in an arm chair during the examination. He talked with considerable freedom, although occasionally at a loss for a word, and especially for a proper name or substantive. He was able to flex the thigh on the pelvis and the leg on the thigh, and with a little effort to carry the paralyzed limb over the thigh of the opposite side. He had a slight movement of extension and flexion of the toes, could dorsally flex the foot in a feeble manner, but could neither abduct nor adduct it, nor could he lift the heel with the calf muscles. All the movements which had returned were comparatively feeble, but the proximal were relatively much stronger than the distal movements of the limb. He was able to extend the forearm somewhat strongly, could feebly flex the forearm at the elbow and also draw the arm backwards. He could pronate the hand when it had been placed in a supine position. He had not at this time recovered any of the movements of the hand and fingers except that of pronation of the hand. The movements regained were all feeble, extension of the forearm which was the first to return being the strongest. Carefully testing for cutaneous sensation it was found that tactile sense was much impaired and that the senses of pain and temperature were also diminished, although these were relatively better preserved than the sense of touch. If anything, cutaneous sensibility was a little more defective at this time than prior to the operation. His hand was still too much paralyzed to test him for the stereognostic sense, but he was tested for muscular sensation and sense of position by blindfolding him and having him put his unparalyzed hand on the right. This he failed to do on his first attempt, but succeeded on later efforts, although he did not accomplish the act readily; he sometimes carried his left hand to the right forearm and then slipped it down to the hand. Evidently the muscle sense was still much impaired. Wasting was still very manifest in the right upper extremity, and especially in the right hand. The right knee-jerk was marked, moderate ankle-clonus was present. The Babinski reflex was absent, although the normal plantar response was not prompt. He had no headache, but complained of some dizziness.

It would be tedious and in the main unprofitable to con-



tinue to trace in great detail the progress of this case. Slow but sure improvement went on from day to day. The leg and arm continued to gain in power and control; sensation in all its forms gradually returned; speech improved until it was normal; and after a proper correction of his refraction had been made by Dr. Risley the patient was able to read about as usual. He continued to have recurring attacks of depression and emotionality, the tendency of these attacks being apparently in part temperamental and in part the result of his long period of apprehension and real suffering. Notes of his condition were made at frequent intervals, but I shall only recall a few of these made at intervals between December 11 and March 3, 1900, on which day he started with his son for a trip of six weeks or more, during which he expected to visit Gibraltar, Genoa, Naples and Alexandria.

On December 18 it was found that he had recovered several additional movements of his leg. He could dorsally flex the foot with facility and also lift the heel from the floor. All the movements formerly regained were greatly improved. He made a good effort to stand and walk with assistance. The arm also showed a decided improvement. He was able to lift the semiflexed arm to the shoulder line. He could also slightly flex the fingers and pronate and supinate the hand. He talked with comparative fluency, only very occasionally being at loss for a word. He read aloud a newspaper extract for me, making only a single mistake. His general health and strength and his mental state were greatly improved.

On December 27 touch, pain and temperature sensations were almost normal; he merely recognized a very slight difference in the degree in which he felt objects in favor of the left side. His hand and arm had gradually regained almost all their movements, although many of these were still feeble, especially those of the hand and fingers. Although scarcely able to grasp objects as small as a knife or key, he was able to recognize them by their shape, but not as promptly or as accurately as with the other hand. His stereognostic sense had returned largely but not fully. He was now able to walk a few steps alone. On December 31 it was noted that the patient could now walk across the room without aid. Movements of the fingers, hand, forearm and arm were still improving. Within a few days some tremor became noticeable in the hand or fingers when he made forcible movements or tried to make such movements of the hand and arm. This tremor frequently recurred, but as his limb grew stronger became less marked. On February 7 the following note was made: I saw the patient last evening and I have seen him at short intervals, but

at least once a week, for several months past. He has been at Atlantic City for four to five weeks, and has continued to improve steadily. He can now walk several blocks and his arm and hand have improved to such an extent that he can make some use of them in feeding himself, putting on his clothing, etc. Yesterday he wrote a short but entirely legible note with pen and ink. Cutaneous sensibility is practically normal. He has gained flesh and his general appearance is excellent. He complains at times, particularly after he has used the limb considerably, of a feeling of heaviness in the right leg, in which muscular sensibility is still somewhat impaired. He also makes somewhat frequent complaints of pain in the head, which he refers to the anterior portion of the cicatrix or wound left by the operation.

Three days before he left for his sea trip I again made a careful examination, and could not discover that he had gone back in a single particular, in spite of the fact that he was at times apprehensive and complained considerably of pain in the head which seemed to me to be due to nervousness and possibly to some sensitiveness of the scalp and membranes left by the operation. The pain at times disappeared entirely and was influenced by the weather and emotional states. His lower extremity retained all the movements that it had gained, so that he was able to stand and walk with facility, both on the level and even up and down stairs, although he complained that the limb became heavy after moderate exercise. Both ataxia and paralysis had largely disappeared from his upper extremity. Cutaneous sensibility was practically normal.

It is difficult accurately to fix the date of the pathological beginning of this case. It appears from the history that the patient was first supposed to be suffering from some affection of the brain in 1884, that in fact he went to Europe on the advice of his physician because of headache and other somewhat indefinite but probably neurasthenic symptoms. After a stay abroad of about two years, however, he seems to have regained his usual health, and it was not until 1894 that he was again seriously troubled. During this year he began to have peculiar paresthetic attacks, which in the light of subsequent developments were evidently a form of limited cerebral sensory discharge. The real time of the beginning of the cerebral lesion was probably a few months, or at the most a year or two prior to the first of these attacks, although it is of course possible that some organic disease may have been present as

early as 1884. It is more reasonable to presume that these earlier symptoms were of a hystero-neurasthenic, or at least of a functional character. With the exception of the occasional subjective sensory attacks in the right arm he had no active localizing symptoms for five years, although he was more or less neurasthenic, hysterical and despondent. The history of the case and the lesion and conditions found at the operation show conclusively that the tumor was subcortical in origin, and it was not until it had been growing for five years and had reached the cortex in the parietal region that persistent active symptoms were manifested. In discussing this case it is of great importance to note the manner in which the objective localizing symptoms developed. These were at first confined to impairment of muscular and cutaneous sensibility with associated astereognosis and ataxia. All other localizing signs, trophic, motor, visual, aphasic and reflex, were practically absent—a fact which, taken in connection with the early focal diagnosis which was made, supports the contention of those who believe that for practical purposes the cerebral areas for sensation and motion can be separated. The later symptoms, in so far as they were organic, were partly those of destruction and partly those of pressure. Unless the early symptoms were kept constantly in mind and a close analysis of the progress of the case from stage to stage was always before one studying its phenomena, the added symptoms serve to cloud rather than to clear the diagnosis.

The two things chiefly relied upon in making a diagnosis were the order of development of the clinical phenomena and a limited number of positive localizing symptoms. It will not be necessary to recall here the facts showing the method in which the case developed step by step after the first appearance of active symptoms in 1899. The appearance and progressive increase of the ataxia and astereognosis, the slow augmentation of clinical phenomena which pointed to pressure or implication of the cerebral motor regions, the increase in amnesia and paraphasia, the presence with variations in degree of ankle-clonus and altered plantar reflex on the same side, and the appearance of decided atrophy, constituted together an orderly train of phenomena which could hardly be explained except on

the ground of an organic focal lesion, and as an enlarging neoplasm could best explain the sequence of events the diagnosis of a cerebral tumor seemed to me inevitable. The positive symptoms were those already so fully detailed as pointing to the parietal lobe.

The most important of the general symptoms of brain tumor were either absent or so subordinated to other features of the case as to not give to them the high rank which they usually attain in the diagnosis of brain tumor. While the patient frequently complained of feelings of pressure or discomfort, and sometimes of localized headache, the pain in the head was not of the agonizing and persistent character, and had not the paroxysms of extreme exacerbation which are such notable features in the majority of cases. The complaints of pain often seemed to be conditioned by the emotional state of the patient. Optic neuritis was again and again sought for, but in vain, and at the very last the eye-ground presented a practically normal appearance, or at the most one that would be explained by refraction disorder. Cerebral nausea and vomiting were not present, although occasionally the patient suffered from temporary disorders of digestion. Vertigo was so infrequent as to receive little or no attention in making up the diagnostic specification. Local spasms and general convulsions were absent. From the standpoint of general symptoms the case might have been one of hysteria or neurasthenia plus disorder of refraction.

It cannot be doubted that much in the history of the case and many of the symptoms pointed to the diagnosis of grave hysteria. Among these symptoms were unilateral hypesthesia to touch, pain and temperature; persistent contraction of the fields for form and color, irregular color reversals, and frequently recurring states of emotional depression and excitement. When to these were added the fact that the ataxia and paresis vacillated considerably until a late period before the operation, and that optic neuritis and the other general symptoms of brain tumor were absent or subordinate, it will be seen that many reasons existed either for the diagnosis of hysteria or for holding in balance the diagnosis between organic and functional disease. This case affords a striking illustration of

the importance of not making the diagnosis of hysteria until every probability of organic disease has been carefully excluded, and also brings into the foreground the fact so well known to every experienced neurologist that a hysterical syndrome is so often associated with the phenomena of organic lesion. It cannot be too strongly emphasized that a large majority of the cases of brain tumor suffer sooner or later from hysteria. In this case I do not incline to the opinion that the sensory symptoms were hysterical in nature, but rather that they were due to the localization of the lesion, but it is nevertheless true that hysterical stigmata, sensory, motor, visceral and psychical, may be present in any serious organic case.

As is well known, the writer has long maintained that for all practical purposes of diagnosis and of treatment the cerebral areas of cutaneous and muscular sensibility are separate from the motor area, and that the former surround the latter on the lateral and mesal aspects of the hemisphere; in other words, that they are situated in the gyrus fornicatus, quadrate lobule and parietal convolutions. It would be a useless waste of time again to bring up the reasons for and against this view. A few recorded cases besides those of the writer would seem to indicate that not only is this position justified as regards a general area for cutaneous sensibility, but also that in special portions of the sensory area subareas of representation of different portions of the body may be located. The cutaneous hypesthesia in this case persisted until the operation and many weeks subsequently. The thalamus was not directly involved in the lesion, although of course it is possible that it may have been affected by pressure. The tumor was partly cortical and partly subcortical, being chiefly situated in the corona radiata, where it may have involved or compressed the tracts for cutaneous sensation passing between the thalamus and the cortex, but tumors similarly situated in the acknowledged motor area, cortical and subcortical, do not as a rule, or even with any frequency, cause similar persisting sensory symptoms. The most positive and interesting localizing symptoms in the case were disorders of muscular sense with ataxia. This case, with that of Starr and McCosh,<sup>1</sup> furnish striking evidence in favor of the

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<sup>1</sup> Am. Jour. Med. Sci., Nov., 1894.

view that the muscular sense is represented in the postero-parietal region. In the case of Starr and McCosh, as will be recalled, a small angioma with some surrounding brain tissue was removed by operation at a point about the junction of the superior and inferior parietal convolutions, clearly caudad of the post-central convolution, with the result of producing complete loss of the muscular sense in the opposite hand and wrist with ataxia, the symptoms disappearing a few weeks after the operation. The results of a surgical lesion in this case were identical with those produced by the tumor of the parietal lobule in the case here reported, the only difference being that in the case of Starr and McCosh the symptoms disappeared, while in my case they were persistent and were added to as the neoplasm increased in size and invaded more and more of the adjacent cerebral substance. True motor paralysis and other evidences of implication of the motor cortex or pyramidal tract were absent until the neoplasm by pressure and invasion had extended beyond the cerebral areas for the muscular sense. It would seem that this case is almost conclusive in its teachings with regard to the separate localization of a region for muscular sensibility.

As the case progressed some atrophy was evident both in the upper and lower extremities, but more marked in the upper, and especially in the forearm and hand. On November 19, when the consultation was held with Drs. Osler and Lloyd, this atrophy was especially studied, and as previously noted the muscles of the hand, forearm, arm, shoulder, breast and leg showed distinct atrophy. At one time it was generally taught in the text-books that when hemiplegia came on after adult life has been reached muscular atrophy of the paralyzed side did not follow, but it is of course now well known that real atrophy, not merely the wasting from disuse, is present in a considerable percentage of hemiplegic cases. Atrophy was present in thirty-five of a series of sixty cases studied in the neurological wards of the Philadelphia Hospital a few years since. While the degree of atrophy in some of these cases was slight, in others it was so marked as to be easily represented in a photograph of the patient. These atrophies are usually attributed to degeneration of the pyramidal tracts and involutions of the

ventral horns, and to degenerations subsequent to peripheral neuritis occurring in the paralyzed limbs; but it is not improbable that in some of the most marked cases the atrophy is related to the destruction of special trophic centers in the cerebrum. Savill<sup>2</sup> has recorded a case similar in some of its features to the one here reported, a case in which a destructive lesion of the gyrus fornicatus produced persistent vasomotor and trophic changes in addition to a marked but temporary loss of cutaneous sensation. It is worthy of remark that the lesion in the case here recorded extended beneath the cortex very close to the median surface of the hemisphere, and probably involved to a certain extent the subcortical substance in the same position as the lesion in Savill's patient. Before the operation I suggested to Dr. Keen the probability that the tumor had invaded or nearly reached the cortex of the mesal aspect of the hemisphere in the region of the quadrate lobule or the posterior extremity of the gyrus fornicatus, and this was the case as nearly as could be determined by the operation. While the true trophic centers are protuberant and spinal, lesions in certain regions of the cerebrum may induce atrophy by acting from a distance on these centers. These cerebral trophic centers are probably not to be sought for, as has been suggested by some, in the motor region of the cortex, but in the limbic and parietal lobes, which were the chief seats of lesion in the present case and in that of Savill.

As the marked amnesia and paraphasia disappeared in a comparatively short time after the operation, it is probable that they were due to pressure upon the concept areas or upon the tracts which associate the receptive areas for speech with those on the motor side of the brain; or it may be that the amnesia and speech disturbance, like the emotional condition of the patient, were due to the irritating and inhibitory effects of the tumor, and to the general demoralization of the patient.

The concentric limitation of the fields of vision and the presence on one or two occasions of temporary hemianopsia have their explanation in the pressure of the tumor upon the angular gyre and optic radiations.

The normal plantar reflex for at least two months before

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<sup>2</sup> Brain, V., 14, 1891.

the operation could not be promptly and fully elicited on the affected as on the other side, and the Babinski reflex was present towards the last. Doubtless variations in the degree of pressure on the pyramidal tracts accounted for vacillations in the plantar response which became more and more abnormal as the pressure on the pyramidal tract increased, and not improbably just before the operation some fibers of this tract were destructively implicated in the lesion. The same remarks are applicable in explanation of the variations in the knee-jerk and of the presence of ankle-clonus.

#### SURGICAL REPORT BY DR. KEEN.

Operation Nov. 24, 1899. The location of the tumor, as determined by Dr. Mills and the other neurologists in the case, was deemed to be in the superior parietal lobule, probably subcortical and probably impinging on the median surface, and either directly involving or producing pressure upon the upper part of the post-Rolandic convolution. In thinking over the operation, it occurred to me that it would be a very serious difficulty if I encountered a thick skull, as the flap was not to be in the squamous portion of the temporal, but the parietal, and that it might require a very long time to chisel a large flap in a bone of unknown thickness, and it might even be impossible to fracture the base of the osteoplastic flap. This difficulty I knew had occurred to some of my surgical friends and very seriously complicated the operation. Accordingly, having located the fissure of Rolando, I outlined by my eye the large flap I proposed to make. At one of the two points at the base between which I wished to fracture the flap I made a trephine opening 0.5 cm. in diameter and found that the skull was unusually thick, one centimeter. Had I not first trephined the flap, and so determined its thickness, I should undoubtedly have had great difficulty. A second trephine opening was then made at the other point, and a Gigli wire saw was passed between them. The bone was then sawn about half through without, as was discovered afterwards, producing any injury to the brain. The flap was then outlined by the knife and the bony flap chiseled through. Each of the three sides of the flap measured 10 cm. long. The superior side was parallel



with the median line at a distance of one centimeter. The anterior border was just in front of the fissure of Rolando. No serious difficulty was encountered other than the time consumed on account of the thickness of the skull. This time was diminished also by the freedom with which I chiseled, knowing definitely the thickness of the skull. The dura bulged moderately into the opening. It did not seem more resistant than usual at the upper part, but at the lower portion the dura felt distinctly softer and suggested that there was probably fluid beneath it.

In order to avoid the large veins, and especially their parasinoidal enlargements, the dura was incised with the base of the dural flap above. As I was dividing the dura along the anterior border, the tumor suddenly came into view at the anterior-superior angle of the opening.

When removed, I may add that the tumor measured 5.5 cm. by 4.5 cm., and weighed one ounce and three drams. It was made up of small granular masses about the size of those of an ordinary raspberry, and was of a deep red or purple color. In order to expose the entire tumor a considerable portion of bone was removed by the rongeur forceps anteriorly and superiorly, and the opening in the dura was extended until the entire tumor was in sight. No exact localization could be made by fissures, for by the pressure of the tumor all of these had been obliterated. Evidently the tumor had begun as a sub-cortical mass and had recently burst through the cortex, for at the margins the cortical substance overlapped the tumor by wedge-like portions of cortex, and gradually thinned as it approached the tumor. By the knife and scissors I then incised and cut the cerebral substance overlapping the tumor, thus separating it from the brain substance. Toward the posterior and inferior border of the tumor I opened a large cyst lying at a depth of 5 mm. below the surface of the cortex. We estimated the amount of fluid which escaped as fully an ounce and a half and possibly two ounces. Some of it was caught in a sterile bottle and was examined for bacteria by Dr. Joseph Walsh. It proved to be sterile. Immediately upon the evacuation of this fluid the patient's general condition improved in every respect. In order to measure the depth of this cavity

and yet not to break through its wall, as I might if I used such a stiff and heavy body as a probe or a pair of hemostatic forceps, I took a thread of silkworm gut, bent it double like a hairpin and passed it into the cyst. When I had inserted it 10 cm. I reached the limit. I introduced my little finger, which is 6.5 cm. long, without reaching the extremity of the sac. This maneuver gave me the additional information that I could get the finger underneath the tumor. The tip of the finger was almost exactly against the falx cerebri. The cavity of the cyst was lined with a whitish membrane covered with soft granulation tissue. Three large veins which led into the tumor were ligated with catgut passed somewhat deeply into the cerebral tissue by a Hagedorn needle, and the tumor was then entirely detached from the cerebral substance. I then found that the walls of the cyst were fairly strong, and with scissors, Horsley's blunt knife and my finger nail I was able to detach the entire cyst from the adjacent cerebral tissue, and gradually to draw it out. No hemorrhage of any amount followed its removal. Packing with iodoform gauze checked the oozing in a few minutes. The opening in the dura was closed with catgut without drainage and it is worth noting perhaps that the cavity left by the tumor and the cyst by this time had filled up so completely that the cerebral tissues were in contact with the closed dura. The flap was then sutured a little iodoform gauze being introduced between the bone and the dura at one point.

Surgically the patient made a complete and very smooth recovery. The day after the operation his temperature rose to 101.4°. By the fourth day it had reached the normal, after which there was no rise of temperature. The stitches were all removed on the sixth day, the wound being entirely well.

Dr. Mills has stated the other facts in reference to his recovery.

On February 23, just three months after the operation, he came to my hospital, got out of a cab unaided, walked up the steps with a just perceptible limp, and when I came into the reception room he jumped up with what might be termed alacrity from his chair, came forward and stretching out his right hand shook hands with me. His speech was entirely

normal, his mental condition excellent. A week later he started on a pleasure trip to the Mediterranean and Egypt.

Remarks.—I must confess that while I was enucleating the sac I was in constant expectation of tearing open the ventricle, but happily, in spite of the great penetration of the tumor, 10 cm. (four inches) by measure, no such accident occurred. In view of his extremely bad physical and mental condition at the time of operation, it is especially gratifying that his recovery at the end of so short a time as three months was so complete. He was practically in every respect his former self, saving a very slight limp, which would scarcely attract attention except from one who was familiar with his history.

PATHOLOGICAL REPORT BY DR. SPILLER.

From the Wm. Pepper Clinical Laboratory (Phœbe A. Hearst Foundation).

The tumor was very dark in color, vascular, round and almost globular. A cyst was beneath the tumor, and the wall of this cyst was thick and had therefore been removed at the operation without rupture.

The tumor consists of numerous cells with large round single nuclei staining deeply with Delafield's hematoxylin, and in some places contains small hemorrhages. Blood vessels are very numerous, and about many of these a layer of columnar cells is found. Sections from the interior of the tumor resemble sections from a large round cell sarcoma, but more stroma is present than is found in sarcomas. In portions of the tumor which are of more recent origin the appearance is like that of an endothelioma. Distinct bands of cells containing large round nuclei, occasionally two nuclei, with much intervening tissue are found in these portions, and the bands of cells are not separated by open spaces from the surrounding tissue. A blood vessel is seen in the center of some of these columns of cells, and the cells are arranged about the vessel in such a way that they appear to have originated from it. The tumor is not encapsulated, and is not sharply defined from the surrounding cerebral tissue, and at some places the invasion of the cerebral tissue by columns of tumor cells is distinctly seen.

A portion of the cortex adjoining the tumor was studied

by the thionin stain. Many of the nerve cells near the tumor are greatly atrophied, are deeply stained, and have apical processes twisted like a corkscrew. In some cells the cell body is not thicker than the apical process. All the nerve cells stain diffusely, and chromophilic elements in many of the cells are absent, even in the larger cells where normally they are distinct.



Nerve cells of the cortex near the tumor, greatly atrophied, deeply stained, and having apical processes like a corkscrew.

I would class the tumor as a perithelioma or—as many object to the name of perithelioma—as an endothelioma. The growth probably originated in the walls of the blood vessels, and certainly did not arise in the dura, as the dura was not even adherent to the tumor.

## A CASE OF AMAUROTIC FAMILY IDIOCY.\*

By HUGH T. PATRICK, M.D., Chicago.

In 1896,<sup>1</sup> Dr. B. Sachs proposed the name "amaurotic family idiocy" for a peculiar disease previously described under various titles, principally by oculists, the first case having been recorded by Warren Tay in 1881.<sup>2</sup> In 1887,<sup>3</sup> Sachs himself reported the first case with autopsy and a little later Kingdon<sup>4</sup> reported a case, also with autopsy, and first called attention to the similarity of the cases reported by the ophthalmologists and those described by Sachs, the latter having recorded another case in 1892.<sup>5</sup>

One of the best pathological reports is that of Hirsch,<sup>6</sup> who made a careful histological investigation of all the principal parts of the nervous system. To this report was added an unusually competent microscopic examination of the eyes by Doctor Holden.<sup>7</sup> Hirsch was able to collect twenty-six cases, and at the same meeting of the American Neurological Association to which he reported his case Frederick Peterson<sup>8</sup> added one more, also with autopsy and careful microscopic examination.

So far as I have been able to ascertain no important additions have been made to the clinical picture of the disease since the article by Sachs in 1896. The principal features are briefly as follows: Semitic, generally eastern Hebrew, parentage with no evidence of inherited syphilis or other diathesis; nearly always more than one child in the same family attacked by the disease; normal birth at full term and normal development for a few months, followed by rather rapid physical and mental failure, together with loss of vision and peculiar fundus changes, the child dying of marasmus at about the age of two

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\*Read before the Chicago Neurological Society, Dec. 4, 1899.

<sup>1</sup> New York Medical Journal, May 30, 1896.

<sup>2</sup> Transactions of Ophthalmological Society of United Kingdom.

<sup>3</sup> JOURNAL OF NERVOUS AND MENTAL DISEASE, 1887, p. 541.

<sup>4</sup> Ophthalmological Society Transactions (England), Vol. XII.

<sup>5</sup> JOURNAL OF NERVOUS AND MENTAL DISEASE, 1892, p. 603.

<sup>6</sup> *Ibid.*, July, 1898, p. 538.

<sup>7</sup> *Ibid.*, p. 550.

<sup>8</sup> *Ibid.*, July, 1898, p. 529.

years. There may or may not be nystagmus, strabismus, rigidity or contractures and the reflexes may be diminished or exaggerated. In a large proportion of the patients abnormal sensitiveness to noise is present, or at least an exaggerated reaction to auditory impressions. The fundus appearance is pathognomonic of this disease and is present in all cases, for which reason I have induced Dr. Beard to make some of his beautiful paintings of the fundus in my own and Dr. Kuh's cases. My case is briefly as follows:

H. M., male, was first seen March 2, 1899, at which time he was fifteen months old. He was the first child, there having been no previous miscarriages, was born at full term to young and healthy parents who are not Jews and are not blood relatives. The labor was normal, lasted twelve hours, was not difficult, and the child was not asphyxiated at birth. So far as can be learned from the parents he developed in a perfectly normal way until the age of nine months, never had convulsions or illness of any kind, and was nursed by the mother for nearly a year. At nine months the child was able to sit alone, but from this time gradually failed in strength until when seen by me, six months after the onset, he could hold neither the trunk nor head erect and was unable to creep. The appearance and nutrition were very fair, the head was of normal size and well formed, there were no signs of rickets, syphilis or tuberculosis, but in addition to the general failure of strength there were general spasticity, excess of associated movement and a distinct athetoid character in voluntary movements, such as are often seen in moderately severe cerebral diplegias. Evidently the child was also deficient mentally, as he was apathetic, although somewhat restless, and understood practically nothing, but could occasionally be made to smile in a silly way. Indeed, the mother volunteered the information that he was not as bright as formerly. The deep reflexes were exaggerated and upon any exertion or marked sensory impression there was a very noticeable tendency of the extremities to stiffen. As is at once seen from the foregoing, the case looked not unlike one of bilateral infantile cerebral paralysis with imbecility or idiocy, but in addition to the history of normal birth, of normal development until the age of nine months, and of gradual inception and slow progression of the disease, the presence of amblyopia, also of gradual production, and the extreme susceptibility of the child to noises, caused me to make a diagnosis of amaurotic family idiocy which was fully sustained by immediate ophthalmoscopic ex-

amination. There was still some vision, as the child noticed a candle flame and, two or three times, apparently saw movements of the hands between the eyes and a good light. The pupils were equal and reacted to light. Occasionally there were transitory lateral nystagmoid movements of the eyes, and quite frequently a slower rolling about of the eyeballs, most frequently upward. The abnormal susceptibility of the child to noise deserves special mention, as it was well marked and, as before noted, is frequent in this disease. It has been recorded as hyperacusis without, so far as I can ascertain, any evidence that the sense of hearing was abnormally acute. The condition is rather that of undue reaction to sound, or exaggeration of the general auditory reflex, and it is worthy of note that these children as a rule also react excessively to ordinary sensory stimuli. In my case a gentle slap caused a great start which at once passed into a tonic contraction of the entire body, particularly manifest in the extremities. A light tap on the patellar tendon for the purpose of eliciting the knee-jerk had the same effect. As before mentioned, these excessive reactions are not unusual in cerebral diplegias, but in my patient the slamming of a door would cause a generalized rigidity so excessive as to almost merit the name of spasm. From this time until death at the age of one year and ten months the disease ran a typical course. Within a few weeks of the time of my first examination the amblyopia had progressed to amaurosis with loss of pupillary reflex to light, and the atrophic changes in the optic disc continued to increase. The child lost flesh, and finally became completely paralyzed. The unnatural reaction to noises and sensory impressions continued to the end. For two weeks before death the child vomited at intervals, and during the last few days, judging from statements of the mother and attending physician, presented a picture resembling that of acute meningitis. That is, there were high temperature, violent action of the heart, extreme hyperesthesia to noise and touch, extreme retraction of the head and strabismus. There was no rigor mortis.

For some unascertained reason the family physician discouraged the making of a post-mortem examination, and I was not informed of the child's death until nearly two months later.

The only striking peculiarity of my case was the absence of Semitic parentage.

## A CASE OF AMAUROTIC FAMILY IDIOCY.

By SYDNEY KUH, M.D., Chicago.

The case which I am about to report differs in some essential points from most of the other cases of amaurotic family idiocy heretofore recorded, so that I should have had some hesitation about making the diagnosis had it not been for the pathognomonic changes in the fundus oculi.

The patient, J. E., 13 months old, was brought to my office for examination on the 22nd of October, 1898. Her parents are Jewish, the father is a clerk. The family history is absolutely negative. There is no consanguinity, no alcoholism, no history of nervous or mental disease in the ancestry. The father denies having ever had any symptoms of syphilis. The little girl was the only child, born at full term, and the labor was neither protracted nor unusually difficult, no instruments having been used. During pregnancy the mother's health was good; she suffered no trauma.

The little girl seemed to develop normally until she reached the age of five months; she was breast-fed. At the time just mentioned the mother first noticed that the child could no longer hold up her head. At about the same time the patient's vision began to suffer, though she still seems to notice light. She moves the hands and legs but has very little strength in either, cannot sit up without support, never learned to stand or walk, has made no attempt to talk, does not seem to know her mother any more or to see anything. When spoken to the child will turn towards the source of sound. The hearing appears to be good. At the age of one year our little patient had her first tooth. Obstinate constipation. During the last two months the patient has suffered from spasms which are rapidly increasing in frequency. Since the little girl was six months old she has hardly ever cried.

*Status præsens:* The child is about normal in size, but very much emaciated. The muscles are all soft and flabby, except in the right arm, in which there is some rigidity; are atrophied all over the body. The facial expression is that of an idiot. The skull is large. The fontanelles are still open. The ears are unusually large, the lobules adherent. The child has a saddle nose. Dr. Haight was kind enough to make a rhinological examination for me, the result of which was negative. The pupils are wide and react to light. Any sudden noise, such as the clapping of hands, slamming of a door, etc., causes generalized spasm (hyperacuity). At times it seemed as though the child noticed a bright light, but we are not cer-



tain about this; objects that were not very bright were surely not seen. The deep reflexes are absent. The internal organs appear normal; there is no swelling of glands; no sign of skin disease.

During the examination the child had a number of spasms, partly due to some noise and then general, partly spontaneous and then limited to certain parts of the body. First the left side of the face began to twitch and both eyes were turned to the left, then there was a series of rhythmical contractions of the extrinsic muscles of the eyes, causing the eyeballs to move from the central line to the left. Shortly after this the convulsions affected the right arm and leg and a little later the left leg became similarly affected.

Ord.: 0.1 each of iodide of potassium and bromide of strontium *t. i. d.*

On November 12 the child was again brought for examination. The mother said that the spasms had become less frequent and less violent and lasted only a shorter time. During the last week there had been hardly any. To-day, however, they are again more frequent. Medicine had not been given regularly. During the last days the child has cried frequently again. Has had seven teeth within the last three weeks. The appetite is good and patient looks as though she had gained some in weight. The pupils are dilated. Pulse-rate 132 a minute. Patient seems brighter. Clapping of the hands, which formerly always produced spasms, has no such effect now; but there are irregular clonic spasms, affecting various parts of the body, during which the eyeballs are generally turned to the left. They seem to arise spontaneously. After that examination the patient was not brought to the office again, but on April 16, 1899, Drs. Haight and Beard and I visited her at her residence. We then found the skull greatly enlarged, broad in the parietal regions and with arched frontal bones. It measured  $21\frac{3}{8}$  inches in circumference. The spasms are decidedly less frequent, resemble more the petit mal type. The general condition is about the same as at the time of the previous examination; the child now has all of her teeth.

A few days ago I wished to see the little girl again and upon visiting her father heard that she had died three months ago. The paresis had gradually increased until there was total paralysis of all four extremities; the child became more emaciated while the skull increased in size until it measured  $22\frac{1}{2}$  inches shortly before the patient's death. The spasms had not become more frequent again.

The points in which this case differs from those reported by

others are, firstly, the great frequency of the epileptic spasms and, secondly, the existence of a pronounced hydrocephalus. While both of these symptoms have been recorded in some other instances, they do not appear to have been as prominent features as they were in my case.

Sachs' theory that amaurotic family idiocy is due to arrested development of nervous tissues seems to me not in accordance with the facts as presented by my case. There appeared to be something more, a distinctly retrogressive and destructive change. A perusal of the observations made by others tends only to strengthen that conviction.

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#### THE APPEARANCES OF THE FUNDUS OCULI.

BY CHAS. H. BEARD, M.D.

I must express my gratitude to Drs. Kuh and Patrick for having afforded me the opportunity of studying ophthalmoscopically these two interesting cases and of making a colored drawing in each. The ophthalmoscopic pictures here presented are unique. There is no other in any way similar. Not even that seen in embolism of the central artery of the retina, as has been more than once asserted. When it comes to appearances of the fundus oculi *characteristic* of a general disease or disorder, these stand alone, "grand, gloomy and peculiar." To describe them briefly:

The disc is of normal size and stands out unusually clear-cut as to its borders, and the choroidal ring is specially distinct—*i. e.* free from the obscuring radiate striation normally present in the eyes of children. The lamina cribrosa is slightly veiled, though visible. Contrary to what I had been led to expect, there is not pronounced atrophy of the optic nerve and retina. The outer half of the disc is decidedly blanched, and the retinal vessels are all somewhat reduced in size. The choroid and the hexagonal pigment, wherever visible, appear normal. The great dominating feature of the picture, and that which characterizes this fundus and is absolutely diagnostic of the disease in question, is what one sees in and immediately around the macula lutea. Surrounding the fovea centralis, and concentric with it, though fully two or three times as large, is a

liver-colored disc. This disc is the center of a zone of grayish-white, which extends for at least two discs diameters horizontally, and somewhat less vertically in every direction from the center, and gradually fades away into the normal red-orange of the eyeground. This livid disc is as clear-cut as a coin—not irregular in outline, as is the case in acute inflammatory conditions where the surrounding retina is infiltrated; nor is it either cherry-red or carmine, as in those other instances, but is distinctly brownish. Instead of coinciding with the fovea in area, as is stated above, it is larger. That is to say, instead of marking the area which is occupied by the cones alone it marks that which is devoid of the ganglion cells.

Another highly distinguishing feature is observed in the character of the whitish zone surrounding the center. This is nebulous rather than cloudy. It is nearly white at the circumference of the liver-colored disc, thence gradually thins away to nothing, but is translucent and shows *some* color throughout. Far from obscuring the retinal vessels which enter it, it only serves to make them more distinct by contrast, so that one is able to trace the tiniest of them right up to the central spot.

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- 46 UN CAS D'AMYOTROPHIE PROGRESSIVE D'ORIGINE TRAUMATIQUE (A Case of Progressive Amyotrophy of Traumatic Origin). F. Sano (Journal de Neurologie, 1899, No. 23, p. 441).

Sano reports the case of a man who was severely injured in the back and thorax. About two months and a half after the accident the right lower limb began to waste and sensation in the part became diminished. Reaction of degeneration was not obtained. The wasting of the limb may have been due to neuritis, although the absence of pain on pressure over the nerves, and of vasomotor and trophic disturbances of the skin, and of disturbance of sensation limited to the distribution of certain nerves, made the diagnosis of progressive atrophy of spinal origin seem more probable. A few cases of spinal atrophy resulting from trauma have been reported. SPILLER.

- 47 STÖRUNGEN DER VASOMOTORENTHÄTIGKEIT UND DER SENSIBILITÄT NACH PERIPHERER TRAUMATISCHER FACIALISLÄHMUNG (Disturbance of the Vasomotor Functions and of Sensation Following Peripheral Traumatic Facial Paralysis). Carl Biehl (Wiener klin. Wochenschrift, 13, 1900, p. 131).

Biehl reports a case in which the left facial nerve was injured by a stab-wound in front of the left ear. The sensation in the distribution of this nerve was much impaired, and perspiration over the left cheek and reddening of this cheek occurred when the patient ate. As the fifth nerve was not injured, Biehl thinks the case proves that the facial nerve contains vasomotor and sensory fibers. SPILLER.

## RAYNAUD'S DISEASE IN THE INSANE.

By J. E. COURTNEY, M.D.,

First Assistant Physician of the Hudson River State Hospital, Poughkeepsie, N. Y.

Attacks of superficial symmetrical gangrene, or Raynaud's disease, are more common among the insane than among people suffering from nervous disorders in which there is no mental disease proper. It might naturally be anticipated that vasomotor and trophic disorders being so common in the insane would often take this form, but articles on insanity have paid the subject little attention. Senile dementias, and terminal dementias at all ages, show the condition oftenest, but it may be observed in other types of insanity. Sometimes the attacks are abortive; small areas appear in the extremities, oftener involving the toes or the fingers, in which there is pallor, coldness and slight anesthesia which gradually disappear without death of any portion, or the tips of the fingers or back of the hand may darken, the skin shrink and peel, resembling severe sunburn; the process, however, closing short of gangrene. The morbid process may be more decided, there being a shallow but actual gangrene of some portions of the extremities, the skin finally becoming detached, and the raw surface gradually healing. It is striking that the loss of tissue is nothing like as extensive as the blackening of the surface in the early stages of the attack would indicate, and predictions as to the extent of injury likely to result cannot safely be based on it. More women than men among the insane are attacked.

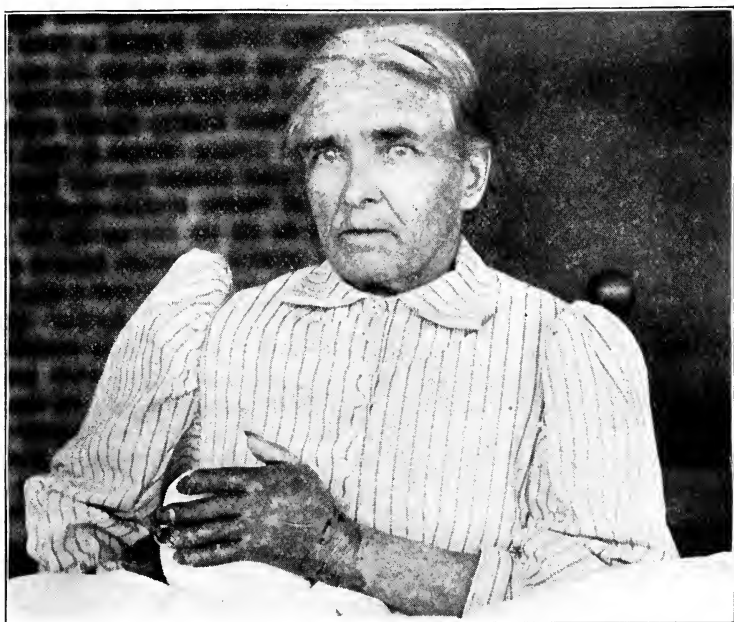
The trouble is the result of lowered vitality and lack of vasomotor adjustment.

There are several other conditions seen in the insane which must be closely allied to this both clinically and pathologically. I refer to onychias, hypertrophies and loss of the nails. These states are common and result from stasis and severe local depression of the circulation.

The following case of Raynaud's disease in an insane woman has recently come under my observation:

Kate G., age 60, intemperate, was found to be suffering prematurely from senile dementia. Both feet and the left hand

were red and slightly swollen. On the dorsal surface of each foot, just above the toes, was a rounded granulating surface about one inch in diameter; the entire left hand to a well-defined line a trifle above the wrist (see fig.) was denuded of skin and presented an indolent ulcerating surface with patches of new skin beginning to form. At the flexure of the wrist there were crevices in the flesh. The patient said that the attack began with numbness and blackening of the skin, which gradually became detached "by the aid of poultices." In the medi-



cal history submitted the item about the hand read "left hand diseased, refused examination, has appearance of gangrene." Some of the pulp of the thumb and index finger was gone, and the nails were twisted and elongated. The feet soon healed entirely and the hand is now natural in appearance as regards the skin, but the joints are quite stiff and the nails thick.

The treatment of Raynaud's disease as I have seen it among the insane is a matter of elevating the nutritive functions, rest, hygiene and tonics generally, and locally warmth to limit the injury and afterwards scrupulous cleanliness.

## TWO CASES OF MUSCULAR ATROPHY OF THE PERONEAL TYPE.

BY GIVEN CAMPBELL, M.D.

The rarity with which this type of muscular atrophy occurs, the lack of agreement among neurologists as to its underlying pathological basis, and the advanced age at which my case commences, renders it one of special interest; nor is the interest lessened because of the fact that my patient is a physician, an intelligent and trained observer, able to give a very competent account of the symptoms at a time when the history of such cases is usually incomplete. The following is the history as taken from my case book:

L. C. N., male, aged 45 years, applied for treatment October 5, 1899.

Family history: Patient has several sisters and one brother. All of these are in good health and show no signs of a myopathic heredity. Mother and father healthy. Mother had six brothers and four sisters. These are all healthy. One brother has a daughter that developed epilepsy at puberty. One sister is the mother of nine healthy daughters, all unmarried, and of one son now ten years old. This boy (first cousin to my patient) did not begin to walk until  $2\frac{1}{2}$  years old, and at that time it was noticed that his lower legs were wasted and weak. This wasting and weakness have progressed in both legs, being accompanied by fibrillary twitching. This condition was not preceded by pseudo-hypertrophy, but led to distinct talipes varo-equinus, for which an operation was done a few weeks ago. I have not been able to see this boy, but his mother states that he is slightly saddle-backed and that his upper extremities are not weak.

Past history: He never had any serious illness or injury. Five years ago he had a right sciatica with pain and tenderness along the course of the nerve trunk in the thigh. This lasted five days and was not severe. Again twenty months ago, and four months after onset of present disease, he had a second attack of sciatica. This also was located in the right limb and caused pain and tenderness along the nerve trunk in the thigh, and had a mild course and duration of five days. He has had no acute infection for the last twenty years, and has never had syphilis. He was a moderate drinker for ten years prior to onset of present malady, but has drunk nothing alcoholic since. During this time he averaged one glass of whiskey and two glasses of beer daily, though he never went on sprees. He was unusually susceptible to effects of alcohol. He was never

exposed to the action of lead and was moderate in the use of tobacco.

**Symptoms:** Up to September, 1897, he was well, and led the active life of a country practitioner. At this time, however, he noticed that he turned his ankles very easily, that his thigh muscles showed an unusual tendency to cramp, and that he was not able to lift his toe properly in walking. He began to wear out the toe of his shoe. He noticed a wasting of his anterior tibial muscles and tested his knee-jerk and found it absent. He is sure, however, that there was no considerable weakness in the extensors of the knee at this time. Thinking that he had tabes, he tested himself carefully for the presence of any sensory symptoms, but failed to find any. His sensibility to touch, pain and temperature was normal, and he had no ataxia. He was able to stand on one foot with his eyes closed. A few weeks later these tests were all verified by a brother physician, and again no sensory symptoms were found, but the knee-jerk was still absent even with reinforcement. The weakness and wasting continued to increase in the legs symmetrically and was gradually progressive. By the end of five or six months the anterior tibial group was almost as atrophic as at present, and the muscles of the anterior aspect of the thigh were beginning to be affected. About this time the patient noticed twitchings in the gluteal muscles and the lumbar group. These twitchings have persisted ever since, and are severe enough to be troublesome to him. They occur during sleep, and have been present in the shoulder girdle muscles for the last year. They are most severe in the right arm (biceps and deltoid). This twitching is worse when he has been agitated or is a little run down. The disease has gradually progressed in the lower limbs so that they are now quite useless, and he can barely move the toes in flexion. No other movement is possible. He states that his sexual functions have been normal up to one month ago, but that since then there has been a slight falling off in venereal desire, and at present date, December 5, this decrease has progressed to a complete loss of all desire. Sphincters are normal. He has had no sensory involvement of any kind at any time in the course of the disease, although this was carefully and frequently searched for by an observer expecting to find it.

**Physical examination:** He is of healthy aspect and fairly well nourished. His face shows no signs of muscular atrophy, his eyes are normal, he has no nystagmus, and his pupillary reflex is normal. An examination of the fundus was made by Dr. Wolfner and revealed nothing abnormal. Vision is normal. No signs of any cranial nerve lesion are present.

Upper extremities: No distinct signs of muscular atrophy are present, but both deltoids seem shorter than normal. The small muscles of hand are neither atrophic nor weak. Grasp: right hand 115, left 93. The left deltoid is distinctly weak. He can hold his right arm out from the shoulder, supporting a weight of seven and three-quarter pounds. Considering that he uses his arms a great deal in wheeling himself about in an invalid chair, he is not strong, even in the right deltoid. With the left arm he can support under similar conditions only two and seven-eighths pounds. The left supinator longus is only about one-third as strong as the corresponding muscle on the right side. It is interesting to note that the twitchings, which are distinctly visible now and are of a coarse fibrillary nature, are chiefly located in the right arm, the one less paralyzed. Body muscles: Thoracic muscles are normal; the lumbar group is not atrophic; the abdominal muscles seem perhaps somewhat atrophic. Lower limbs: The atrophy is marked in both lower extremities. Perhaps the adductors are rather less atrophic than the others. The lower leg contains very little if any muscular tissue. The limbs are perfectly helpless.

Reflexes: The deep reflexes are everywhere absent. Cremasteric reflex is present.

Electrical reaction: Some twitching is seen in the quadriceps muscles of the lower limbs, but these muscles fail to react to either galvanism or faradism. In the upper limbs, including left deltoid and supinator, the reactions to galvanism and faradism are normal. No reaction of degeneration and no sensory impairment can be found. Some shortening of calf muscles is present, so that the foot cannot be brought quite to a right angle with the bone of lower leg. Attempts to bend it farther cause pain referred to the belly of calf muscles.

The pulse now varies between 85 and 100, but the patient states that it was always frequent. The viscera are normal and the urine shows nothing abnormal. He has no tenderness along the nerve trunks.

The most interesting points in regard to this case are: 1st. The advanced age at which the disease began, forty-three years. I can find no record of a case beginning this late. 2d. The rather rapid progress of the disease. 3d. The distinct weakness of the left deltoid and the supinator of that side, muscles not usually affected in this disease, and the occurrence of this weakness before there is any perceptible atrophy of the hand or forearm. 4th. The complete absence of sensory



symptoms. 5th. The abolishment of the knee-jerk at a time when the extensors of the knee were apparently in perfect condition and when no sensory disturbance was present. The fibrillary twitching was permanent and quite constant; it was distinctly visible, and was perhaps the symptom pointing most strongly to an involvement of the spinal cord. Were it not for this and the tendency to cramps of the muscles the absence of all sensory impairment might make this case seem one in which the muscles were primarily involved. The question naturally arises as to why the deep reflexes were absent when the voluntary muscular power was good. The fact that no sensory disturbance has ever occurred would seem to imply that the defect was not on the afferent side of the reflex arc, notwithstanding the autopsy findings by Marinesco and by Sainton, in whose cases a degeneration of the posterior columns with changes in the cells of the anterior horns was found. But in Marinesco's case a distinct sensory involvement was present. In Sainton's case it was not excluded. In investigating the efferent side of the arc, we find a good condition of psychomotor impulses, and this would imply that the muscle, motor plate and efferent neuron were normal up to their connections with the pyramidal tract.

The writer is well aware that Paul Sainton<sup>1</sup> has attempted to exclude from the category of Charcot-Marie amyotrophy cases in which the hands are not atrophied and in which the shoulder girdle muscles are involved. It is also plain that in my case the progress of the disease has been more rapid than is perhaps the rule, and that the atrophy does not follow the garter type in the lower limbs to which Charcot first called attention. The clinical picture is one of weakness and wasting going hand in hand, perfectly symmetrical, and of insidious progress, commencing in the peroneal group of muscles and extending upward, accompanied by no sensory impairment, with early loss of knee-jerk, complicated by distinct fibrillary twitchings and by the tendency of the muscles to cramp. This picture, taken in connection with the fact of a similar malady occurring in a first cousin, leaves room for no other diagnosis

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<sup>1</sup> Thèse du Doctorat, 1899.

than that of a muscular atrophy of the peroneal type, to which the French have given the name of its discoverers, Charcot and Marie, and the English that of its principal British exponent, Howard Tooth. I might remark that Tooth<sup>2</sup> has reported several cases in which the upper extremities were not involved. Sachs's<sup>3</sup> case was one in which there was no wasting of the hands, but there was wasting of the entire thighs and some slight wasting of the infraspinatus muscles. In Jacoby's<sup>4</sup> case the upper extremities were not affected, and some lordosis was present.

If the consideration of this case brings out any especial points of interest they are:

1st. That muscular atrophy of the peroneal type may commence so late in life as forty-three years.

2d. That the atrophy is not always of the garter type in the lower limbs (extending a little above the knee), but may involve the entire thigh.

3d. That well-marked cases may occur in which the hands are not atrophic.

4th. That the knee-jerks may be early abolished in cases which are, and subsequently remain, free from all sensory impairment.

Since reporting the above a second case of this disease has been referred to me for treatment. My two patients are in no way related to each other and live in different parts of the country. The clinical history of this second patient is as follows:

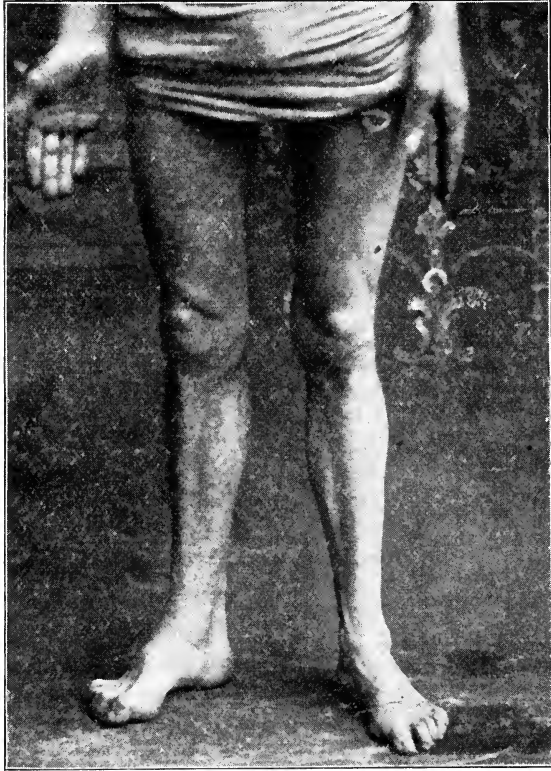
W. K., male, aged 32, height 5 feet 10 inches. Family history: Mother's sister epileptic. Otherwise family history is unimportant. Past history: Patient was born at term after normal gestation and labor. Well until 2 years of age. Nursed on breast by mother until 1 year old, but mother's milk was deficient in quantity and deficiency was made up by oatmeal water, this being given from time of birth. At 2 years of age patient had an attack of epidemic meningitis, one month's duration, good recovery, no sequelæ. Patient was a perfectly healthy and active boy until 15 years of age, at which time his mother noticed that his instep was gradually becom-

<sup>2</sup> Thesis Cambridge, 1880.

<sup>3</sup> Brain, Jan., 1890.

<sup>4</sup> JOURNAL OF NERVOUS AND MENTAL DISEASE, 1894.

ing higher. She had to have shoes made to order for him. This deformity has steadily progressed, the feet becoming smaller and the instep more arched. Progress gradual and steady. For several years after onset of the atrophy there was very little inconvenience in walking. This has increased lately, and is due rather to the deformity than to any muscular weakness. Six years ago patient began to be



Case II.—The deformity of claw-foot is especially well shown on the right side in the photograph.

much troubled by cramps, chiefly in the muscles of the legs. This tendency, while not so severe, is still distinctly present, and can be elicited at will by patient forcibly contracting flexors of knee or calf muscles. During the last four years distinct fibrillary twitchings have been present, principally in the muscles of the arms.

There has never been any pain or other sensory involvement. Physical health has remained good. For the last fifteen years he has been gradually growing deaf, and this loss of hearing has been complete for the past three years. Several years ago he noticed a dimness of vision, for which he consulted an oculist, by whom a diagnosis of double stellate capsular cataract was made. Vision is now very imperfect. Rectal and vesical symptoms are absent. Sexual power is normal. Patient is moderate in the use of tobacco and does not use alcohol, tea or coffee.

Physical examination reveals no disease of the vital organs. Urinary secretion normal. The muscle reflexes are very greatly diminished. Knee-jerks barely obtainable by reinforcement. The superficial reflexes, however, are distinctly increased, tickling the sole of the foot almost producing a clonic spasm of the flexors of knee. Sensation is normal to touch, pain and temperature. No tenderness of the nerve trunks is present. Measurements: Calf of leg, right, 31.5 cm., left, 31.5 cm.; thigh 5 cm. above upper margin of patella, right, 32 cm., left, 31 cm.; thigh 7.5 cm. below gluteal fold, right, 46 cm., left, 46 cm. The neck measures 34.5 cm. Electrical reactions normal. There is a slight but perceptible wasting of the small muscles of the hands. The small muscles of the feet are very atrophic, and the deformity of claw-foot is very distinct and pronounced. The small size of the lower leg is probably due to disuse rather than to the direct effects of the disease, for it will be noticed that the entire limb, excepting the foot, is symmetrically small.

A comparison of this case with the one first reported will be of interest in considering the rapidity of onset in this disease. Both cases are extreme, the first being unusually rapid in its march and the second very slow; for after a period of seventeen years the atrophy has not extended noticeably beyond the small muscles of the feet and hands. The existence of cataract and deafness is probably a mere coincidence, although the fact that the deafness is total and involves bony conduction makes it probable that it is of central origin.

## NEW YORK NEUROLOGICAL SOCIETY.

March 6, 1900.

The Vice-President, Dr. Joseph Collins, in the chair.

### ANEURISM OF AORTIC ARCH; PUPILLARY ABNORMALITY.

Dr. W. B. Noyes presented a man, forty-six years of age, who had had the initial lesion of syphilis four years ago. He had been referred to Dr. Noyes recently for a recurrent laryngeal paralysis, the result of pressure of an aneurism of the arch of the aorta. Further examination had shown increased knee-jerk, mental symptoms, a pupillary abnormality, and a mental dulness which suggested an incipient general paresis. The left pupil was contracted; the knee-jerks were markedly increased; under any mental strain he showed twitching of the face. The case was exhibited to determine whether or not it might be possible for such an aneurism to cause this pupillary phenomenon by interference with the sympathetic branches. The aneurism was not sufficiently large to involve the spinal cord.

Dr. G. M. Hammond said that he had made a superficial examination of this man. There could be no question about the existence of certain mental defects, and from a consideration of all the signs and symptoms it seemed to him probable that the man was suffering from cerebral syphilis. This was ample to account for the change in voice and for the other symptoms.

Dr. Pearce Bailey was disposed to explain the alteration in the voice and the pupil by pressure on the recurrent laryngeal nerve and interference with the sympathetic.

Dr. B. Sachs said that the very great difference in the two radial pulses seemed sufficient evidence of the presence of the aneurism, and the aphonia was characteristic of peripheral disturbance resulting from solid neoplasm or an aneurism. The condition was entirely typical of what was commonly found in general paresis, cerebro-spinal syphilis and some other conditions. The mental symptoms did not seem to him characteristic of an ordinary general paresis.

Dr. W. M. Leszynsky said that both pupils reacted very well to convergence, showing that in all probability the pupillary reaction was not dependent upon any special syphilitic condition.

Dr. Noyes said that the examination of the eyes showed mixed astigmatism in both eyes, slight paleness of both optic discs, and weakness of the eye muscles, but no definite paralysis. Laryngoscopic examination showed plainly a recurrent laryngeal paralysis. It had existed about three weeks, and was improving under anti-syphilitic treatment.

### SPECIFIC CERVICAL PACHYMEMINGITIS.

Dr. Sachs presented a man, thirty-eight years of age, who gave a fair history of having had a hemiplegic attack about three years ago. From this he had recovered promptly, and had had no further trouble until last October. At this time he

had developed a complete rigidity of the cervico-dorsal region of the spine. In addition, he had complained of great pain radiating from the lower cervical region into the arm, and had presented a very marked atrophy of the muscles of the right shoulder girdle. A diagnosis had been made of specific pachymeningitis of the cervical region. The case was interesting as presenting a clear example of extra-spinal syphilis. The pains and atrophy had been interpreted as root symptoms. There was a clear history of syphilitic infection having taken place five years ago. The patient had been put upon increasing doses of the iodide, and had taken as much as 60 drops of the saturated solution three times a day. He was now entirely free from pain, and could move his head very freely. The speaker said that, in his experience, these cases had been extremely rare.

Dr. Joseph Fraenkel said that there was at present a similar case at the Montefiore Home. The patient was a man, about thirty-two years of age, who gave no history of syphilis. He had had about four months ago a transient hemiplegia, with some mental deterioration. Six weeks later he had developed pain and stiffness in the neck. He had been admitted about two weeks ago, and at that time had exhibited a marked torticollis. He was stupid and apathetic; the pupils were unequal and did not react to light. There were evidences of the old right-sided hemiplegia, and on the left lateral portion of his neck were two distinct and rather hard tumefactions. The trapezius, deltoid, biceps, triceps and the interdigital muscles on the left side were all considerably atrophied, and gave the reaction of degeneration. All the muscles on the left side of the neck were in a state of increased myotatic irritability. The diagnosis had been made of a syphilitic meningitis of the base, and of the upper part of the cord. Under anti-syphilitic treatment improvement had been quite rapid.

Dr. Joseph Collins thought there could be no doubt that the case just presented was one of those rare examples of syphilitic pachymeningitis, rare in that it attacked the roots. He had seen the case referred to by the last speaker. There had been a true myositis in the cervical region, producing a torticollis, muscular atrophy and degeneration. He saw no reason for the statement that this case was a parallel to the one presented by Dr. Sachs.

#### DISEASE OF THE THORACIC DIVISION OF THE SYMPATHETIC CHAIN.

Dr. Joseph Fraenkel read a paper with this title. He said that most of the text-books made meagre reference to the sympathetic system, and, indeed, there was very little known about the thoracic sympathetic. The following case was reported: The patient was a woman, and she was first seen on July 25, 1898. Her family history was excellent, and she herself was of simple and temperate habits. She had been in good health up to ten days prior to this first examination. At that time she had awakened at midnight with a peculiar pain

in the left upper extremity, and this had soon been followed by painful contractions of this extremity, and finally of the other extremities. The attack had lasted a few minutes and had terminated in a laughing spell. No aura had been detected. The objective findings, after an exhaustive examination, had justified only a diagnosis of hysteria. On April 17, 1899, he had found her sick in bed, complaining chiefly of severe pain in the thoracic region. The rhythm of the pulse was greatly disturbed, but otherwise the physical examination was negative. Subsequently there had been a rise of temperature, and auscultation had then revealed a distinct systolic murmur over the heart, which was attributed to the great distention of the abdomen with gas and the consequent displacement of the heart. The next morning she had been very much better, but the following evening she had been in collapse. She died before a consultation with a surgeon could be held. At the autopsy, made by Dr. Van Gieson, the four upper sympathetic ganglia on both sides were found buried in a mass of purulent material. The pus lay beneath the pleura, near the vertebral column. There were no evidences of recent reactive inflammation in the neighborhood of this abscess. The other findings were negative. Staphylococci and streptococci were found in the pus. There were no tubercle bacilli present. The speaker expressed the opinion that hysteria was the only possible clinical diagnosis in this case, and it should be remembered that fatal hysteria was not unheard of. He had been able to collect fourteen such cases. The case raised the interesting question as to whether the closing symptoms were connected with the earlier ones, or were entirely independent of them, and due to the accidental involvement of the thoracic sympathetic ganglia.

Dr. Hammond said that the report did not seem to him to connect the lesions found at autopsy with the symptoms detailed in the clinical history. The inference was, that the patient at first had hysteria, and had subsequently developed the abscess which had caused her death.

Dr. Sachs said he was pleased to learn that there were very few recorded cases of fatal hysteria that could stand a critical examination. He had himself had at one time under his care a woman of twenty-five, who had presented all possible hysterical phenomena. The diagnosis of hysteria had been made by himself and by others, and had been persisted in until the woman suddenly developed coma. This had been followed rather suddenly by anuria and death, and the autopsy revealed chronic nephritis. Of course, the latter had had nothing to do with the hysteria. In the case under discussion some of the later symptoms that had been attributed to the hysteria had been really the result of the abscess of the pleura. It would seem to him that this abscess must have also involved some of the roots as they emerge from the vertebral column; hence the symptoms presented could hardly be interpreted as having been entirely the result

of an invasion of the sympathetic chain. The case certainly did not present any marked relation between hysteria and the thoracic sympathetic.

Dr. Leszynsky said that hysteria is a functional nervous disorder which occasionally results in organic disease, and so indirectly might be the cause of death. It was well for the general practitioner to remember that every symptom observed in a hysterical subject is not necessarily a part of the hysteria.

Dr. Collins took the ground that the first symptoms presented in this report were those of traumatic hysteria, and the later symptoms were dependent upon the formation of a subpleural abscess. The woman unquestionably had not had hysteria until after her marriage, yet hysteria, like epilepsy, was a psychosis of early life. The abscess had apparently been of very slow formation, and quite circumscribed near the very last, and a very small focus of degeneration could easily have caused her symptoms. The case seemed to show the possibility of the occurrence of hysteria as a part of derangement of the sympathetic.

Dr. Fraenkel said that he had endeavored to report in detail, and without prejudice, this case, which was clinically hysteria beyond all question. It was generally acknowledged that hysteria may occasionally cause death, usually through some paralysis of the sympathetic. The slight lesion of the sympathetic found at this autopsy would have been undoubtedly overlooked had the pathologist not been put on his guard by the request that a special search be made into the condition of the sympathetic.

#### TORTICOLLIS AND ITS TREATMENT.

Dr. W. P. Wilkin reported a case of torticollis occurring in a journalist thirty-three years old. He had enjoyed good health previously, was of a nervous temperament, and lived liberally. In the summer of 1898 his work had been exceptionally arduous, and he had then become neurasthenic. After a time he had begun to suffer from an aching feeling in the back of the neck and along the right sterno-mastoid muscle. This had gradually developed into a marked torticollis. The man had entered St. Luke's Hospital in May, 1899, and had been made to rest in bed for all but two hours out of the twenty-four, and a support had been applied. Various methods of external and internal treatment had been tried without benefit. He had then been treated by the speaker at the Post-Graduate Hospital by suspension, and by the internal use of eighteen-grain doses of the valerianate of zinc. A point of importance in the treatment seemed to be allowing him considerable freedom in the hospital and a liberal diet, and placing him in a private room instead of in a hospital ward. Subsequently the man had experienced a great deal of relief while in a London hospital from the use of one grain of exalgin three times daily. In these spasmodic cases gentle pressure with one or two fingers was often enough to restore the head after it had been drawn violently to one side. One of the best methods of



treatment for this variety seemed to be an abundance of rest and a quiet outdoor life. The employment of drugs alone could hardly be expected to accomplish much good.

Dr. Collins remarked that he had seen a number of cases of torticollis, but never one so severe and so disabling as this one. It was marvelous to see the prompt improvement that had followed the use of suspension.

Dr. Hammond said that a number of years ago he had formulated the hypothesis that clonic torticollis was a result of irritation of the nuclei of the nerves, and that tonic torticollis was the result of irritation of the nerves themselves. Later experience had not led him to change this view, although he was aware that others did not accept this theory. From the history of the case just reported it seemed that this case had had at least a partial psychical origin. The beneficial influence of suspension would lead him to suspect a psychical origin, for there was no reason for expecting benefit to follow suspension in a case of organic torticollis. In his own practice he had secured the best results from conium when pushed to its physiological limits. It had often proved serviceable in the organic cases when other remedies had failed. In the psychical cases the selection of the remedy was not of much importance unless the psychical impression could be relieved. In the case under discussion he could not help feeling that the means used had been entirely inadequate to accomplish the result unless the case had been largely psychical.

Dr. Leszynsky said that he had been intending to report two cases which had come under his care during the past six years and both of which had recovered. One of these patients, a woman, had been kept in bed from two to six months at a time. In addition to the general management so well understood, these patients had received massage and gymnastic manipulations of the head and neck. The first patient had had this systematic treatment for a period of nine months. In his case suspension had seemed to act both psychically and mechanically. The attendant had been instructed to use massage up to the point of thoroughly stretching and exhausting the muscle. In both of these cases he had used atropin, conium, and other remedies without much satisfaction. Suspension would cause stretching of the muscle, and would in this way give it the needed rest.

Dr. R. G. Wiener said that he had had four cases of torticollis which he had been able to observe for a long time. Two of these patients were males with an alcoholic history. By the hypodermic injection directly into the muscle of atropin and morphin in increasing doses, these patients had been thoroughly restored in nine and eleven weeks respectively. The third case had been treated surgically but had relapsed. The fourth case had proved rebellious to all treatment.

#### A CASE PRESENTING CURIOUS HYSTERICAL DISTURBANCES OF VISION.

Dr. Phillip Meirowitz reported this case, which occurred in a woman of twenty-five, of neurotic temperament and inheritance. In the spring of 1895 she had begun to suffer from hysterical attacks. Subsequently she had developed laughing and crying spells, and these had become more frequent and severe. When seen in March, 1898, the seizures had not been so severe, and consisted chiefly of crying, brought on by

mental strain. Frequently, when gazing into the air, or when looking at the pavement, colored concentric rings would be seen. In darkened corners she would see certain peculiar figures of various colors and shapes. On rising from a couch she frequently saw a succession of blue commas surrounded by green spots. These would disappear on looking out of the window, but people passing in the street would be surrounded by colored borders. Often the images were duplicated and showed the complementary color. At times her memory became distressingly bad, and the spelling of words became difficult. Her condition had been ameliorated by hypnotism. When first seen in June, 1896, hysterical stigmata had been sought for, but not discovered. Examination of the eyes showed hysterical astigmatism.

Dr. Leszynsky said it was not unusual for the more intelligent hysterical patients to describe a great variety and multiplicity of symptoms. It was difficult to determine how much was purely subjective.

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- 48 NÉVRALGIE PARESTHÉSIQUE SUR UN MEMBRE ATTEINT DE PARALYSIE INFANTILE ANCIENNE (Paresthetic Neuralgia in a Limb Paralyzed from an Old Infantile Palsy). Paul Sollier (*Journal de Neurologie*, January 20, 1900, p. 21).

The peculiar form of sensory disturbance known as paresthetic meralgia has been seen only in the distribution of the external cutaneous nerve of the thigh; there is no reason, however, why it should not occur in the distribution of other cutaneous nerves, and Sollier reports such a case. He speaks of this sensory disturbance as paresthetic *neuralgia*. A man of fifty-three years had an imperfectly developed lower limb as a result of infantile paralysis. Within the last eighteen months he suffered from sciatica in this limb. The pain was felt to some extent in the posterior part of the thigh, but its chief location was in the external part of the leg. The foot was swollen and painful. Pressure over the sciatic caused no pain. The urine was normal. The postero-external part of the leg and the external part of the dorsal surface of the foot were anesthetic, and electrical sensibility was considerably diminished in this area. The man also had paresthesia in this region. As the limb was useless on account of the infantile paralysis, resection of the external popliteal nerve was performed, and four months later the patient wrote that he was cured of his paresthetic neuralgia. The portion of nerve resected was examined by Nageotte and found to be diseased. SPILLER.

## PHILADELPHIA NEUROLOGICAL SOCIETY.

January 22, 1900.

The President, Dr. James Hendrie Lloyd, in the chair.

### A CASE OF HEMIPLEGIA WITH PARTIAL CLAW-HAND.

Dr. A. Ferree Witmer exhibited a man who in May, 1896, had fallen unconscious and had remained unconscious for three days. He was then found to be paralyzed on the left side, but was able to walk about six weeks after the "insult." In February, 1899, he fractured the left forearm near the wrist. The man was presented because he had the unusual combination of left hemiplegia with contracture of the extensors of the left fingers. Objective sensation and the electrical reactions were normal. It was possible that the anomalous position of the hand was the result of the fracture.

Dr. Spiller replied to the objection that had been made that the case might be one of hysteria by saying that the patient had been carefully examined at the Polyclinic Hospital, and no evidences of hysteria could be found. The shortening in the extensor muscles of the fingers was shown by the fact that the fingers could be extended when the hand was extended at the wrist, but were drawn backward at the points of insertion of the common extensor when the hand was flexed at the wrist.

Dr. F. X. Dercum presented two cases of primary neurotic atrophy bearing a resemblance to multiple neuritis.

Dr. D. J. McCarthy asked whether any change in the arteries was present. He referred to a case that he had been investigating, where amputation had been done for senile gangrene. He had found distinct arterial sclerosis with a high grade of interstitial neuritis. In the nerves of the leg only one-fifteenth to one-tenth of the usual number of nerve fibers was present. No electrical or sensory examination had been made.

The changes in the nerves that may follow circulatory disturbances are of interest. In some experiments that he had made on dogs by tying the femoral artery no changes were noticed in the anterior crural nerve after five to ten days. This probably was on account of the free anastomosis. If the ligature had been applied to the internal iliac, he thought that changes in the nerve would have been found.

Dr. William G. Spiller said that the correctness of the view of Dr. McCarthy had been shown by the examination of the tissues from cases of arterial disease in man. When sudden closure of an artery occurs the nerves rapidly degenerate, but if the occlusion is gradual the degeneration of nerve fibers is less likely to occur.

Dr. A. A. Eshner asked how the differentiation from multiple neuritis would be made in cases such as Dr. Dercum presented. This differentiation might be easier in the presence than in the absence of sensory phenomena. He asked whether there might not be a peripheral neuritis largely of motor type in which the symptoms would be those of palsy with wasting and other trophic phenomena like those presented by Dr. Dercum's patients.

Dr. F. X. Dercum, in closing the discussion, said that there were

no arterial changes in these cases. The diagnosis between multiple neuritis and primary neurotic atrophy was made largely by the histories of the cases. In multiple neuritis there is an onset which is acute or slow according to the cause, associated with an etiology which is unmistakable. Not only is there foot-drop, but also wrist-drop. The pain also would be different in the two affections. Here the pains have occasionally been sharp, but usually they are of a dull, aching character. There is no tenderness on pressure as in ordinary multiple neuritis.

#### TROPHIC CHANGES IN THE DISTRIBUTION OF THE FIRST BRANCH OF THE FIFTH NERVE FOLLOWING TRAUMATISM.

Dr. D. J. McCarthy presented a girl eighteen years of age, who had come under his observation two years ago suffering from headache, with graying of the eyebrow and eyelashes on the left side. Her family and early personal history were negative. Three years previously she was accidentally struck on the left parietal region with a portion of a brick. After the contusion had disappeared, a perceptible graying of the blond eyelashes and eyebrow was noticed. After several months the outer two-thirds of the eyebrow and the eyelashes on the left side had become perfectly white. At the same time the headache developed and caused her much annoyance; the continual worry about the deformity caused by the whitening of the eyebrow and lashes—she at that time being rather prepossessing in appearance—was probably in part the cause of the headache and of a certain amount of nervous irritability; she noticed that there was anesthesia on the left side of the scalp, and on examination this anesthesia was found to be in the distribution of the first branch of the fifth nerve. Five years after the accident there appeared to be some flattening of the face in the frontal region of the left side; the skin was dry and perspiration was much less marked over the left forehead. The anesthesia to touch and pain still persists and the eyebrow, instead of being perfectly white, has many blond hairs scattered here and there among the white ones.

Dr. Wharton Sinkler said that this case resembled those of supraorbital neuralgia with graying of the hair in the distribution of the nerve, described by Anstie. He was inclined to regard the case as one of traumatic neuralgia rather than neuritis, because the injury involved, primarily, the peripheral branches in the parietal region and the graying occurred in brow and eyelashes. The graying of the hair he considered not an unusual occurrence in migraine of the ophthalmic type and in supraorbital neuralgia.

Dr. F. Savary Pearce referred to a case he had just seen of a boy who had distinct atrophy of the skin of the region that was affected in Dr. McCarthy's case and along the course of the right supra-orbital nerve, following an attack of herpes which occurred about Christmas, 1899. There had been no trauma. The boy also had optic

atrophy as a sequel to aural disease. Dr. Pearce had never before seen a condition of this kind following herpes.

Dr. James Hendrie Lloyd remarked that Dr. McCarthy's case suggested the possibility of such a lesion as hemifacial atrophy resulting from trauma. In a brief examination of the case he had not been able to assure himself that there was any alteration of the bone. There was no distinct line of demarcation as is seen in hemifacial atrophy. He did not think that the soft structures were materially involved. The graying of the hair was an interesting feature.

Dr. D. J. McCarthy said that if this were a case of supraorbital neuralgia he did not see why hypesthesia was present. The patient could stand the stick of a pin in the affected area without inconvenience, and she had a greater tendency to perspiration on the affected side. He thought also that there was some slight flattening of the bone with slight thickening of the skin. He had seen graying of the hair in neuralgia, but in those cases the graying was more in the back portion and side of the head.

In regard to facial hemiatrophy, he mentioned a case of Homén of hemifacial atrophy in which a tumor of the Gasserian ganglion was found.

#### A CASE OF PARESTHETIC MERALGIA WITH TRAUMATIC ETIOLOGY.

Dr. D. J. McCarthy presented a man forty-eight years old, from the nervous clinic of the Polyclinic Hospital who had suffered for several years with an uncomfortable creeping sensation over the anterior and external surface of the right thigh. The left thigh during the past year had also become affected. For nine years he had worn a heavy truss to control a bilateral inguinal hernia. The band of the truss fitted closely to the pelvis and exerted considerable pressure on the external cutaneous nerve, especially during lateral movements of the body. On examination, an extensive surface over the anterior and external part of the thigh, extending to a few inches above the patella, was not only the seat of vague, uncomfortable paresthetic sensations, but was also markedly anesthetic to the strongest faradic current, and to a less degree to pain, changes in temperature and touch. The same condition in less intensity was found in the left thigh.

Dr. William G. Spiller said that in April, 1898,<sup>1</sup> in presenting a patient with paresthetic meralgia to the society, he had suggested that operation might be of benefit. The external cutaneous nerve of the thigh is sensory and resection would only cause loss of sensation in the part supplied by it. He had suggested that stretching of the nerve might answer the purpose. At that time he was not aware that operation had been suggested by anyone. Since then Chipault had operated on three cases with great improvement of the symptoms. Dr. Spiller did not believe that operation would prove beneficial in all cases, and in some, as after resection for trifacial neuritis, the pain would probably return.

Dr. F. X. Dercum said that these cases were not uncommon, although the present case was of interest on account of the etiology.

<sup>1</sup> JOURNAL OF NERVOUS AND MENTAL DISEASE, 1898, p. 736.

He suggested that it might be better to speak of them as neuralgia of the external cutaneous nerve than to give them a distinct name as though they were separate and apart from everything else. It is unfortunate to multiply names in medicine.

#### A CASE OF ATYPICAL TREMOR.

Dr. W. G. Spiller presented a man of sixty-five years who had had tremor of the limbs for more than ten years. Slight tendency to propulsion was present, but no other signs of Parkinson's disease existed. The tremor was not a typical one of paralysis agitans, and could be made to vary in character. The patient's mother had had a tremor, and tremor had developed in his brother and sister late in life. The man was exhibited on account of the difficulty of determining whether the tremor was one of senility, of paralysis agitans, of hysteria or of heredity, although it was probably the tremor of paralysis agitans. It is well known that Charcot denied the existence of tremor due to senility.

Dr. F. X. Dercum said that cases of Parkinson's disease differed very greatly from each other. While in the majority of cases the tremor is fine and of small extent, it is not always so. Cases are sometimes met with in which the tremor is so extensive and so intense as to suggest multiple cerebro-spinal sclerosis. Sometimes slowness of movement and rigidity make their appearance long after the tremor has been established.

Dr. F. Savary Pearce said that this man had come to Dr. Weir Mitchell's clinic recently. He was a "striker" in a blacksmith shop, and was still able to work. The occupation may have had some bearing on the etiology. Dr. Pearce had regarded the case as an atypical one of Parkinson's disease.

Dr. Charles K. Mills thought that there was an unclassified form of organic tremor of which perhaps this was an example. He had seen a number of cases which suggested this hypothesis. He had recently seen, for instance, in a man beyond middle age, a marked unilateral tremor somewhat resembling that observable in the present case. He had also recently seen a second case of a man with an extraordinary tremor in his right arm which was increased under excitement and did not present the specific clinical features of either paralysis agitans or of disseminated sclerosis. It seemed to him more likely that we have an unclassified form of organic tremor than that forms of tremor so persistent should be regarded as hysterical. The case shown by Dr. Spiller seemed to approach more closely to paralysis agitans than to any other well-known type of tremor.

Suggestions with regard to cause, while they do not carry great weight, are sometimes of value. He thought that tremors of this kind might be due to some form of miliary sclerosis or disseminated myelitis implicating the motor cortex.

Dr. D. J. McCarthy remarked that in connection with a case like this one of Dr. Spiller a case of Charcot quoted by Gowers is of interest. An initial spastic paraplegia, hysterical in type, disappeared suddenly and returning under emotional excitement passed ultimately into lateral sclerosis. Dr. McCarthy thought it was not improbable that in the course of time Dr. Spiller's case would pass into a true type of paralysis agitans.

Dr. A. A. Eshner said that the general appearance of this patient,

apart from the history, was strongly suggestive of paralysis agitans. It would not be safe to base a diagnosis upon the character of the movements alone. There are cases in which the tremor is absent, probably in consequence of the rigidity. Whether there is any relation between the degree of movement and the existence or the absence of rigidity it is difficult to say. Dr. Mitchell had called attention to the fact that in paralysis agitans if a sudden extensor movement of one of the fingers were made the tremor would cease. This observation had been confirmed in a great many cases observed at the Infirmary for Nervous Diseases. That which thus takes place with passive movement corresponds with what occurs when the patient undertakes a voluntary act, namely: The movement usually ceases; but if the act is persisted in, the movement will, in a brief time, recur, with perhaps increased vigor; then, when the position of rest is resumed, the movement will again cease temporarily.

Dr. A. Ferree Witmer remarked that there were two points of special interest in this case, the neuropathic disposition and the occupation, and he asked if with this association the tremor might not be one of a neurasthenic type.

Dr. James Hendrie Lloyd said that the case impressed him as being of the senile type of tremor, presenting somewhat the appearance of paralysis agitans. The cases of tremor seen in old age are not strictly like paralysis agitans, although they sometimes closely approach that type. It is a mistake to say that in paralysis agitans the tremor can be stopped by intentional movement. It is merely checked for a moment and then returns.

Dr. Lloyd had under occasional observation a gentleman under sixty years of age, engaged in drafting architectural plans, who had this sort of tremor confined to the right arm. He had had it for ten or twelve years.

In the case presented by Dr. Spiller the fact that several members of the patient's family had exhibited a similar condition suggested that the cases should be classed as instances of senile tremor. He did not regard it as hysterical.

Dr. Lloyd also reported that the case of hysterical rhythmical disorder, which he had shown at the last meeting of the society, had been entirely cured by metallo-therapy. The patient was put in a mild hypnotic condition, and the movement was then transferred to the other side by the use of silver coins pressed firmly on the affected limbs and on the forehead, suggestion being made at the same time. After the movement was once transferred it was but a short step to abolish it entirely.

The day before the girl left the hospital she had a genuine epileptic convulsion, and it was then learned that she had been subject to epilepsy for years. There was no doubt of the fit being epileptic: the patient bit her tongue and frothed at the mouth.

#### A CASE OF RHIZOMELIC SPONDYLOSIS.

Dr. A. A. Eshner reported the case of a man, twenty-four years old, a laborer, with a history of rheumatism, but none of syphilis or excesses. He presented cervico-dorsal kyphosis, with rigidity of the spine and of the large joints of the trunk, impaired mobility, general wasting, heightened reflexes and preserved sensibility. Dr. Eshner thought from the symptoms and the physical signs that there was disease not only of the vertebræ and appendages, but also of the spinal cord. The

want of mobility both in the vertebral column and at the shoulder and hip joints would suggest the existence of some chronic hyperplastic process in these situations. Such wasting as was present was universal, and might be attributed to the general impairment of nutrition, while the pains exhibited no localizing distribution. The irritability of the reflexes must be ascribed to involvement of the lateral columns of the cord, whether in inflammation or degeneration, or secondarily to meningitis or to pressure, it was difficult to state. The history of rheumatism might not be without significance from an etiologic point of view, as it has been thought to play such a rôle in other cases of the kind.

Dr. James Hendrie Lloyd thought that there are several distinct conditions so far described under the name of rhizomelic spondylosis, and still others would probably soon turn up. Von Bechterew's case was one of secondary involvement of the vertebrae, due to a lesion of the spinal cord. The posterior nerve roots and posterior columns were involved.

Marie's case seems to have been entirely different. Distinct ossification of ligaments and cartilages was found, with ankylosis and many of the pathological conditions of arthritis deformans. Dr. Lloyd believed that some of these cases are secondary to spinal lesions and he was disposed to think that the case presented by Dr. Eshner was of that type. It is well known, for instance, that some cord-lesions, such as syringomyelia, induce changes in the spine.

The haste of some writers to get into print with an instance of this very dubious affection is to be deprecated. So far all sorts of affections have been reported as instances of it, even including pachymeningitis. Most of these reports are not accompanied by autopsies, so they have little if any value as evidences of a new pathological condition. It seems that Bechterew and Marie are merely engaged in an animated effort to invent a new disease.

Dr. Charles S. Potts referred to a case of spinal rigidity in which the symptoms of cord involvement were very marked that had come under his observation a few months ago. The symptoms, exclusive of the rigidity, would lead one to suspect amyotrophic lateral sclerosis. The man was forty-five years of age and gave a history of pains in various joints of his body, especially the hip and ankle joints and the spinal column. The whole spinal column was rigid and the head was held bent downward and forward. There was excessive atrophy of the muscles about the shoulder and also atrophy, not so excessive, of all other muscles. Myotatic irritability was increased and fibrillary contractions were observed. The superficial reflexes were present, and tests to elicit the plantar reflex caused dorsal flexion of the toes. The patient had ankle-clonus, biceps-jerk, wrist-jerk, triceps-jerk and jaw-jerk. Sensation was normal. The muscles responded to the faradic current, but a stronger current than normal was required. The ankle joints were much enlarged and the bones appeared to be hypertrophied and their movement was much restricted. The case resembled both types that have been described, *i. e.*, that of von Bechterew and that of Strümpell-Marie.

Dr. F. X. Dercum remarked that this case reminded one of rheumatoid arthritis with marked rigidity of the spine. He thought it extremely probable that under the captions given there were included



a variety of affections differing greatly from each other. This is not improbable when we consider the number of affections in which rigidity of the spine may be found.

#### A CASE OF CEREBRAL DIPLEGIA.

Dr. F. X. Dercum presented a woman of 24 years who had been born without instruments and was apparently normal at birth. When eighteen months old she had a fall which was not followed by any permanent injury. She learned to walk at the usual age and had various diseases of childhood. Her intelligence was good.

When about 5 years old it was noticed that she dragged the left leg in walking and that the left arm hung helpless. Other parts of the body were gradually and successively involved. She became unable to walk and her legs and arms assumed the position of marked rigidity and contracture which are noticeable at present.

The patient presents the symptoms of a marked spastic cerebral diplegia of childhood. Both arms are markedly spastic, contracted and the seat of marked athetosis. The left hand and arm are more profoundly affected than the right. The head is frequently drawn to the right and backward while the face is constantly distorted by frequent recurring athetoid movements. The legs are semiflexed, excessively rigid, almost fixed at the knees, and crossed in adduction, while the feet are rigidly extended. She has been in the nervous ward for several years, and only recently, when again studying her case, Dr. Dercum noticed that she had a marked hemianesthesia to all forms of sensation affecting the left side of the face, neck, trunk and left arm and left leg. The anesthesia was fairly well defined in the middle line, but not as sharply as is frequently seen in hysteria.

It was interesting to add that the Babinski reflex was present upon both sides.

#### A CASE OF UNILATERAL INTERNAL HYDROCEPHALUS.

Dr. W. G. Spiller presented the brain from a boy of fourteen years who had had unilateral internal hydrocephalus. The right cerebral hemisphere was a thin sac filled with fluid. The foramen of Monro was not occluded. Four years before death the boy had been able to walk, but he lost this power and his limbs became paralyzed and contractured. He had had many convulsions. The case was important on account of the extreme bilateral contractures, more especially in the lower limbs, resulting from a unilateral lesion, and on account of the internal hydrocephalus involving only one side of the brain.

## CHICAGO NEUROLOGICAL SOCIETY.

December 4, 1899.

The President, Dr. Richard Dewey, in the chair.

Dr. Patrick and Dr. Kuh reported cases of amaurotic family idiocy. See p. 265 and p. 268.

Dr. C. H. Beard exhibited drawings of the fundus of both patients and said that we often hear of certain fundus appearances as being characteristic of certain diseases, such as albuminuric retinitis and diabetic retinitis; but all forms of acute retinal inflammation may merge, at least as regards their ophthalmoscopic aspect. He knew of no fundus appearance so truly characteristic of a general disease as that of the cases here presented. It was identical in both of them, and, from what he could learn, the other reported cases presented the same ophthalmoscopic picture. As regards the similarity of the appearance in these cases to that of embolism of the central artery, the differentiation is exceedingly easy; no one who has seen the two conditions, or an illustration of them, could possibly confuse the two.

The retinal appearance in amaurotic family idiocy bears out the assertion of Dr. Ward A. Holden, that the disease is attended by degeneration of the ganglion cells of the retina. At the rim of the fovea these cells are piled one upon another to a depth of as many as ten; thence they gradually thin down, as the distance from the center increases, to a depth of two or three. It is the degeneration of these cells which makes the white area surrounding the central cherry-red spot. Dr. Beard was surprised to see the amount of retinal circulation in these eyes. The blood vessels were nearly normal in size and there was decided color in the optic discs. Great difficulty was experienced in making the drawings on account of the constant movement of the eyes; not a distinct nystagmus, but a slow rolling of the eyeballs, chiefly up and down. The liver-colored circle was distinctly larger than the pit of the fovea centralis; nearly as many times larger than this as the disc is larger than the liver-colored spot. In the case of Dr. Patrick's patient the mother said that there was no Jewish blood on either side of the family, but her physiognomy seemed to Dr. Beard to be distinctly Jewish.

"Drug addiction, especially in the medical profession," was the title of a paper read by the President.

Dr. C. B. Burr, of Flint, Michigan, said that he was largely or altogether in accord with the conclusions of Dr. Dewey. One point in the development of the drug habit among physicians Dr. Dewey failed to emphasize, and that was, the familiarity which breeds contempt. The physician learns to be careless with the tools of his trade just as does the engineer or machinist. As regards the chloral habit, in an experience of twenty-one years, Dr. Burr had never come across a victim of this habit. The mention of habitués taking a multiplicity of drugs reminded Dr. Burr of one patient under his care who took a mixture of acetanilid, citrate of caffeine, morphine, strophanthus, digitalis and strychnine, and large quantities of alcoholics. Much to Dr. Burr's surprise, the sudden withdrawal of all these drugs caused very little disturbance, and the patient made a rapid recovery. He had also noticed that morphine habitués who drink recover from the effects of withdrawing the morphine more easily than those who take no liquor.

Dr. Dewey had noticed that patients using a great variety of drugs

usually presented less difficulty in their withdrawal than did those who were addicted to a single drug. Dr. Burr had never seen a case of chloroform habit in which the patients drank the drug, but he had had three physicians under his care who were addicted to its inhalation.

Dr. Henry M. Lyman said that when he was an interne in Bellevue Hospital, of the fourteen men on the resident staff, one drank too much, another not only drank too much, but probably took morphine, and a third smoked opium, afterward becoming also addicted to chloroform and whiskey. Dr. Lyman had seen one instance of the chloral habit, but the case was cured without great difficulty. He had known a number of examples of the cocaine habit among physicians. On the occasion of a visit to an institute for the cure of drug habits, he discovered that everyone of the seven or eight physicians on the staff was addicted to one or several drugs. They all had their cures and relapses, but seemed to have the greatest confidence in the method of cure there practised. Dr. Lyman's experience was that most of these cases relapsed.

Dr. J. J. M. Angear thought that the influence of heredity in these cases had been overestimated, and the statement that hereditary influence is a powerful factor had a very demoralizing effect on these patients. Much more frequent and important was lack of moral stamina or some mental condition, and he thought that it would be well to understand that there is no excuse for the morphine fiend any more than for the thief or defrauder. He believed in the gradual withdrawal plan, but during a term of service as surgeon of a state's prison he had never seen any bad results from sudden withdrawal of drugs, although this was the invariable rule in that institution.

Dr. William E. Dold, Lake Geneva, Wisconsin, said he had under his care during the last few months ten cases of drug habit, nine of whom were physicians, the tenth the wife of a physician. Eight of the men were addicted to morphine and cocaine, one to morphine and chloral. He had come to the conclusion that while a man may give up whiskey or morphine, he will continue cocaine if he has been addicted to it for any length of time. The majority of individuals who acquire drug habits are neurotics. He had met several cases of the chloroform habit.

Dr. Sidney Kuh said he was surprised to hear that so few cases of the chloral habit had been seen by those present, as it seemed to him to be far from rare. He had at present under his care a woman who was formerly addicted to chloral, taking forty-five to sixty grains daily, and who, after the cure of this habit, became addicted to trional. As bearing upon the question of the curability of the cocaine habit, he mentioned the case of a physician who for five years used the drug continually, but after an acute illness, during which he was delirious, found that desire for the drug had entirely disappeared, and he has used none of it since.

## Periscope.

### CLINICAL NEUROLOGY.

- 49 EXPERIMENTELLE UNTERSUCHUNGEN ÜBER DAS FEHLEN DES KNEIPHÄNOMENS BEI HOHER RÜCKENSMARKSVERLETZUNG (Experimental Researches on the Failure of the Knee-jerk in Lesions of the Upper Spinal Cord). A. Margulies (Wiener klinische Rundschau, 1899, xiii, 52, S. 925).

After reviewing the literature of the subject, the author gives the results of his own experiments upon rabbits and dogs.

In some cases he cut the cord at about the fifth or sixth cervical segment, in others by proceeding on a wedge-shaped instrument held over the seventh cervical spine he destroyed both vertebral arch and cord at this level, or exposing the cord in the spinal canal he crushed it with a blunt instrument. He found that in the cases sectioned there was always exaggeration of the knee-jerk, while where the cord was crushed, provided the destruction was complete, the knee-jerk was lost, and remained absent for at least a week, while if the destruction was but partial, though temporarily lost, the knee-jerk returned in a few hours or days. Examination of the lumbar segments and nerve roots showed no alteration in the reflex arc. The author finds himself unable to accept Bastian's theory of the cutting off of the influence of the cerebellum and can propose no explanation for the loss of the knee-reflex except through the action of shock, which term he uses in its broadest sense. The exaggeration or absence of the reflex may be a symptom of prognostic importance, since the later condition indicates a more profound injury to the cord.

ALLEN.

- 50 LE TROPHOEDEME CHRONIQUE HÉRÉDITAIRE (Chronic Hereditary Tropho-Edema). Henry Meige (Nouvelle Icon. de la Salpêtrière, Nov., Dec., 1899).

Under the term *dystrophie oedemateuse héréditaire* the author called attention in 1898 to a condition which had been little observed. In the present article are collected observations on eight members of the same family, men and women, comprising four generations, five of whom are now living, all of them having the same affection—a chronic edema, white, hard and painless, which appeared at the age of puberty and occupied sometimes the feet and legs and sometimes the whole extremities and was bilaterally distributed. Such a definite heredity destroys the possibility that this condition may be classed with the edemas more commonly known. The various diseases causing edema are noted, such as phlebitis, erysipelas, elephantiasis, alcoholic edema, angio-neurotic edema, myxedema, hysterical edema and chronic rheumatic edema. From a consideration of the seven cases of hereditary edema found in literature and the present cases, and the cases of Higier and Milroy, the author believes that he is justified in forming a special class of edemas. This edema is hereditary and familiar, capable of invading progressively the lower extremities, stopping at times at the ankle, the knee or the thigh. It is painless, afebrile, chronic and constant. It does not affect the general health in any way and persists to old age. The author proposes to designate the condition by the term chronic hereditary tropho-edema. No definite pathological explanation of the condition is attempted, but

on account of its appearance at the time of puberty and its resistance to all therapeutic measures the disease is probably to be regarded as a developmental one.

SCHWAB.

- 51 UEBER TELEANGIEKTASIEN MIT UNILATERALER HYPERTROPHIE UND ÜBER KNOCHENVERLÄNGERUNG BEI SPINALER KINDERLÄHMUNG (Concerning Telangiectasia with Unilateral Hypertrophy and Concerning Elongation of the Bones in Infantile Spinal Paralysis). S. Kalischer. (Monatsschrift für Psychiatrie und Neurologie, Vol. 6, p. 431).

A child, three and a half years old, had since birth diffuse angioma or lymphangiectasia on the right breast and abdomen as far as the umbilicus, extending at this point beyond the median line. Both sides of the back were involved and similar areas were found on both hips, right arm, left leg, etc. The right lower extremity was much thickened and lengthened. The right hand was larger than the left, although the right thumb and the right big toe were smaller than those on the left side. The left mammary gland was larger than the right. Muscle and bone seemed to share equally in the hypertrophy. No distinct signs of involvement of the nervous system were seen. Kalischer refers to similar cases in the literature and reports a case of infantile spinal paralysis in which one lower extremity at one time was 2 cm. longer than the other, and three years later had become shorter than the other. He thinks the theory of trophic influence and dystrophy best explains the elongation of the bones observed in a few cases of infantile spinal paralysis.

SPILLER.

- 52 UEBER DEN "FEMORALREFLEX" BEI LEITUNGSSTÖRUNG DES DORSALMARKS (The So-called Femoral Reflex in Interrupted Conductivity of the Dorsal Region of the Spinal Cord). E. Remak (Neurol. Centralbl., 18, 1900, p. 7).

The present contribution is interesting in view of the discussion now going on concerning Babinski's extensor reflex of the toes. Remak, in presenting a case before the Berliner Gesellschaft für Psych. und Nervenkrankheiten, Dec. 11, 1899, recalls to the minds of his hearers the fact that in 1893—before this same society—he demonstrated as a "femoral reflex" one which he was able to produce by irritation of a well-defined zone on the upper anterior aspect of the thigh of a four-year-old child with a transverse myelitis below the 7th dorsal segment. This reflex consisted first of a plantar flexion of the first three toes and then of a slow extension of the knee joint through contraction of the quadriceps extensor. The opinion then expressed by him was that there must be a preformed reflex path which—with increased reflex excitability of the lumbar cord from disturbed cord conductivity in approximately the region between the 8th and 12th dorsal segments—permitted stimulation of the above-designated skin area to bring about reflexes always topographically identical. The quadriceps contraction he believed to be the same sort of thing as the "pseudo-knee phenomenon" described by C. Westphal in 1882 in connection with a case of paraplegia from spondylitis of the lower dorsal vertebræ.

Remak then goes on to say that, in the same year (1893), in a discussion of a paper by W. Koenig, "On the Reflex Phenomena to be Observed on Irritation of the Plantar Surface of the Foot," he reported that he had met with three further cases of femoral reflex, two in spondylitic spastic paraplegia in children and one in spinal

tumor at the level of the 7th dorsal vertebra. In all three cases plantar flexion of the big toe, generally but not so regularly, with the same movement of the other toes and extension of the knee, was observed on stroking the thigh.

He further recalls that apparently simultaneously v. Strümpell had published a number of observations (on paraplegics) in which, by irritation of the sole of the foot and the leg, the ordinary flexion reflex was observed, whereas irritation of the skin of the thigh—generally by stroking with the handle of the percussion-hammer, but at times by pricking or by the application of cold—a contraction of the thigh muscles, particularly the quadriceps and the adductors of the hip, was brought about.

These observations did not agree with his own, in that, in his experience, plantar flexion of the first toe occurred much more commonly than the contraction of the quadriceps femoris; and the matter was of particular interest to him because it concerned cases in which irritation of the sole of the foot always brought about the now much-exploited Babinski plantar reflex of the toes.

Remak then reports a case of a 2½-year-old child with spastic paraplegia from Pott's disease involving the region of the 3d and 4th dorsal vertebrae. There was slight rigidity of the legs, the kneejerks and Achilles phenomenon lively, with ankle-clonus at times. The sensation of the lower extremities seemed diminished. Irritation of the sole of the foot caused extension of the great toe, then dorsal flexion of the foot-joint and flexion of the thigh on the pelvis. On the other hand, stroking of the upper anterior portion of the thigh with the handle of the percussion-hammer or with the head of a pin invariably caused plantar flexion of the first three toes, more rarely an extension of the knee. By stronger irritation flexion of the thigh on the pelvis immediately occurred.

In conclusion, R. states it as his impression that the femoral reflex involves the quadriceps more regularly and to a more marked degree in the lower-lying cord lesions (region of 8th dorsal segment), whereas in higher compression only plantar flexion of the toes is brought out, and that it is not improbable that not only the localization of the break in conduction but also its intensity plays a rôle.

J. W. COURTNEY.

53 THE OCCURRENCE OF DAY-TERRORS IN CHILDREN. G. F. Still (Lancet, Feb. 3, 1900).

Day-terrors in children have received but little attention. Much has been written of night-terrors, as they are common, but little attention has been given to the much less common attacks which occur during the waking moments. The author reports five cases of this condition, occurring in children all under the age of six years. In most of the cases the child suddenly begins to scream, has a terrified look and runs to his mother or attendant, hiding his face, and gives evidence of suffering from great fear. Hallucinations and delusions seem to be absent. Cases are reported which have occurred in older children. In some of these distinct evidences of visual and auditory hallucinations were obtainable. The relation to night-terrors is probably very close. In four of the cases reported night-terrors were, or had been, present and it would seem that the day attacks may be associated with them or replace them; but the two conditions are probably very different in etiology, although the etiology of night-terrors is far from being known. A nervous, excitable temperament is nearly always a factor in the etiology. Rheumatism, either in the child or in the parents, may play some rôle. In most of these chil-

dren minor functional neuroses are evident. Habit spasms, nocturnal enuresis, recurring headaches, nervous diarrheas, are frequent with them. In a large number valvular disease is present. Some observers have claimed that night-terrors, and more particularly day-terrors, are epileptic in their nature, and that the condition is to be considered as a form of *petit-mal*. This is not the view held by the author. He believes that, inasmuch as these patients do not show any tendency to attacks of major epilepsy, and, furthermore, are so readily relieved of their symptoms by appropriate treatment, that the relation to epilepsy must be but very slight, if any relationship at all exists. In some cases there seems but a very narrow line existing between day-terrors and some forms of insanity. The author's summary of impressions—for thus far in the study of this affection it is only possible to have impressions—is that it is a paroxysmal psychoneurosis allied to, but not identical with, epilepsy on the one hand and hysteria and insanity on the other. The prognosis is good. The most important part of treatment is the removal of any exciting cause. Since a chronic intestinal catarrh is apt to be present, it should be relieved. Potatoes, pastry and the sugars are to be excluded from the diet. Thread worms or other intestinal parasites must be removed. Potassium citrate and the bromides are useful, and a regular mode of life with healthful distractions and non-exciting sports will usually aid materially in the treatment. JELLIFFE.

- 54 SUR LES ATROPHIES MUSCULAIRES PROGRESSIVES D'ORIGINE MYELOPATHIQUE (On Progressive Muscular Atrophy of Myelopathic Origin. G. Etienne (Nouvelle Icon. de la Salpêtrière, Sept., Oct., 1899).

G. Etienne reports six cases of muscular atrophy of myelopathic origin. These cases are of interest because they depart in some details from the classic description of this disease, but yet they bear such a general resemblance to one another that they evidently belong to the same group. In a general way progressive muscular atrophy of myelopathic origin, or anterior chronic poliomyelitis, or Duchenne-Aran disease, is characterized clinically by the following six points: 1. Onset in the upper extremities. 2. Fibrillary tremor. 3. Reaction of degeneration. 4. Absence of heredity. 5. Commencement at a late adult age. 6. Chronic evolution, about four or five years. The anomalies observed in these cases were: 1. Rapidity of evolution. 2. Early commencement. 3. Modes of onset. 4. Rare complications, as arthropathies. 5. Presence of heredity. Each of the cases quoted illustrates one of these varieties. In the first case, instead of the usual chronic evolution, that is, from three to six years, the whole process was completed in eighteen months after the first manifestations of the disease. In the second case, instead of the usual age, that is, from forty to sixty years, the patient was twenty-four years old at the time of the first symptoms of the disease, which were fatigue conditions in the small muscles of the hand. This patient had had an infantile paralysis involving the right leg. As a rule, the disease begins in the small muscles of the hand, generally the right. The thenar and the short abductor of the thumb are commonly affected first. In the third patient the deltoid and the periscapular muscles showed the first symptoms. The fourth patient had infantile paralysis in the first year of life. The beginning of the present process was in the paralyzed leg and in the muscles of the forearm. The fifth case showed a hereditary history of myelopathic affections. This is very rare in this form of muscular atrophy, but common in myopathic varieties. The sixth case showed the rare condition of arthropathy

of the shoulder joint, and also various trophic disturbances. In this latter case differential diagnosis between syringomyelia and muscular atrophy had to be made.

Although all of these cases show departures from the classic type of the disease, they are of insufficient importance to be separated into different classes. They all belong pathologically to the same group of diseases. The lesions found post-mortem in cases one and six are probably the foundations of the changes found in all, viz.: the alterations in the large cells of the anterior horn. The etiological factor in the first location of the process is essentially overuse of the muscles. The lesions are not, however, confined to the anterior horns alone. In cases one and five the glosso-laryngeal involvement points to a bulbar process. There exists then a morbid series characterized by muscular progressive myelopathic atrophy, progressive labio-glosso-laryngeal paralysis and progressive nuclear ophthalmoplegia—three types of the same disease characterized pathologically by the same lesions of the nervous apparatus in different stages. Each can exist alone or develop by extension into one of the other types. They are essentially, however, the same disease. SCHWAB.

#### ANATOMY AND PHYSIOLOGY.

- 55 STEHEN ALLE GANGLIENZELLEN MIT DEN BLUTGEFÄSSEN IN DIRECTER VERBINDUNG (Are All Ganglion Cells in Direct Connection with Blood vessels)? Albert Adamkiewicz (Neurol. Centralbl., 18, 1900, p. 2).

The author begins with the following hypothesis: Within the central nervous system the capillary circulation of the arteries belongs to the ganglia and is determined by their number and size.

From the standpoint of physiology he considers such hypothesis fully borne out, and argues thus: There is no organ whose function is not under the control of the nerves and no nerves which are not under the command of ganglion cells. The totality of organs makes up the body, the totality of nervous functions the body's life; hence, ganglion function is life. But life, as the supreme expression of material force, demands for its conservation the maximum quantum of the energizing fuel at the disposal of the body—that is, the nutritive elements or, in other words, blood. As A. did not believe that ganglion cells, not being simple tissue elements but independent organs of the most important and complicated sort, could derive their nutrition in the same elementary fashion as do connective tissue and muscle cells, namely, through a plasma stream which oozes from the finest capillaries of end arteries and makes its way through the tissues, he was led to search anatomically for a special and highly developed apparatus subserving this function.

56 Unfortunately for the complete verification of his theories his anatomical researches had to be limited, in the human, to a certain type of ganglion cells—those of the great intervertebral ganglia of the brachial plexus. These he found not to be bathed in a free plasma which reached them in the same diffused current that it did the other tissue elements. On the contrary they received their nutrient stream through the finest sort of vessels, which branched off from the arterial blood-capillaries and formed a sort of capillaries of the second order—*vasa serosa*—which gave passage to the fluid elements of the blood, but to none of the corporeal. One of these minute vessels would make its way to the ganglion cell, spread out and envelop the cell like a glove, narrow down on the further side into an outlet and then pursue



its course until it again entered an arterial blood capillary. From this inexhaustible nutrient gulf stream—its individual preserve, so to speak—the cell through its entire surface took as much energizing material as it needed for its work, and what it gave back in waste products collected in the vacuole of the so-called nucleus and made its way thence through the channel of the central veins of the ganglion back into the venous circulation.

What this stream means to the ganglion cells will be readily appreciated—the author tells us in a semi-“fairlyland of science” aside—if we consider that they have an individual diameter of only some hundred-thousandths parts of a millimeter and that each is practically immersed in a stream which moves at the furious rate of 0.5 mm. per second, while other tissue elements are so intimately coherent that they offer only one surface to the languid plasma as it makes its snail-like way from arterial to venous capillaries.

For further and less particularized proof of his tenets Adamkiewicz has thus far been compelled to fall back upon physiologico-anatomical experimentation. By injecting the brain of a rabbit with carmine gelatin he found that the chief destination of the blood from the carotids was to the cerebral cortex, the manner of ultimate distribution being as follows: From the pial ramifications of the carotid numerous arterioles make their way into the cortex. These run (in a radiating fashion, quite close together) through the three ganglionic layers—the cortical, nuclear and intermediate layer—(elsewhere described by A.) and stop abruptly just at the boundary between the ganglionic storehouse, so to speak, and the projection layer. These arterioles give off lateral twigs which furnish the interlying ganglionic territory with a rich vascular network. The density of this network is not everywhere equal. The cortical layer (*Rindenschicht*—in A.'s nomenclature) is almost completely wanting in such network and of the other two the nuclear layer (*Kernschicht*), which is the richer in ganglia, is the more vascular.

In this circulatory peculiarity of the rabbit's brain A. sees strong support of his principles concerning the human species, *i. e.*, that the arterial flow to the central nervous organs is to the ganglion cells; and in proof of his proposition that also in the brain the arterial twigs stand in direct, though here not histologically demonstrable, connection with ganglion cells, he offers the following physiological argumentation: Every ganglion possesses a determinate function. If the ganglion is aroused, its function is awakened. If it is continuously irritated, prolonged and pathological functional activity ensues. Irritation of the ganglia of the cerebral cortex, for example, calls forth—as proof of its pathological activity—nystagmus, slowing of the heart's action and the respiration and tetanic spasm of the body muscles. All these phenomena, formerly falsely believed to be the result of “brain pressure,” are, in the author's opinion, simply signs of protest on the part of corporeal organic functions under the control of the brain.

All unphysiological influences are a source of irritation for the ganglion substance as well as for nervous tissue. Even distilled water comes in this category, for if one denudes the brain of calvarium and membranes and allows distilled water to drop from a burette upon the denuded brain, the irrigation may be continued for hours without producing signs of cerebral irritation. One may even directly expose the ganglionic substance of the hemispheres to a forcible stream of water without resulting irritative phenomena, but if the same irritating agent—distilled water—is introduced into the brain through a carotid it requires only two or three centimeters to produce nystagmus, very

marked disturbance of respiration and pulse and tetanic spasm in all the extensors of the body.

Adamkiewicz believes that this difference in the action of the same irritant can be explained only on the ground that when acting through the circulation it comes into more intimate relation with the ganglionic substance than in any other way, and that the logical conclusion from this is that the ganglia must lie within the arterial conduits and be completely immersed in blood.

His ultimate conclusions are as follows: All arteries which enter the brain and spinal cord of man and of animals—at least the higher—end on the yonside of the capillaries in minute plasma vessels, which contain the ganglia cells in diverticula.

J. W. COURTNEY.

- 56 BEITRÄGE ZUR FEINEREN ANATOMIE DER GROSSHIRNRINDE (Contributions to the Finer Anatomy of the Cerebral Cortex). Hans Berger (Monatsschrift für Psychiatrie und Neurologie, Vol. 6, 1899, p. 405).

In an interesting article Berger reports the results of his study of the visual cortex of man. He examined: 1. The cortex of both calcarine fissures of a man sixty-five years old, who had lost the sight of both eyes twenty years previously on account of phthisis bulbi and detached retina, and had died from broncho-pneumonia without having shown evidence of mental disease. The brain of a woman of sixty-three years who had died from rupture of an aneurism of the aorta was used for comparison.

2. The cortex of both calcarine fissures of a woman of thirty-nine years who had been blind in the left eye for fourteen years. The brain of a woman of forty-four years who had died from typhoid fever was used for comparison.

3. The cortex of both calcarine fissures of a patient fifty years old with parietic dementia and complete optic atrophy of eight years' duration. The brain of a laborer of about the same age was used for comparison.

In these cases the optic atrophy was complete or bilateral. His statements are somewhat difficult to understand. In one place he seems to say that distinct variation from the normal was found only in the visual cortex of the man who had been blind for twenty years. Again, he states that in all his cases of blindness a distinct diminution of the cortical cells was seen; but further on he says that no distinct atrophy was found in the case of parietic dementia. In the brain of the woman with unilateral blindness the cells of the occipital cortex opposite to the blind eye were more atrophied than those in the occipital cortex of the same side. This atrophy of the cells of the visual cortex was regarded as similar to that which occurs in the cells of the anterior horns in hemiplegia. The atrophy of the cells extended throughout the cell layers of the visual cortex, and the cells especially concerned with the perception of light could not be distinguished.

SPILLER.

## Book Reviews.

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ORTHOPEDIC SURGERY. By William E. Moore, M.D., Minneapolis, Minn. W. B. Saunders, Philadelphia, 1898.

There is no question but that the accomplished orthopedist should be a good surgeon as well as a good mechanic. In this work the author has kept the idea of surgical interference well in the foreground, yet not to such an extent as to disregard any of the recognized methods of mechanical treatment. He states that it has been his aim to take the middle ground between the surgeon who operates too frequently and the orthopedist who seldom operates. Those methods of treatment only have been described which have stood the test of time, and are advocated by the best members of the profession engaged in this special line of work.

He has stated clearly when and how to operate, and what class of cases would best be benefited by some mechanical appliance. Only the simplest apparatus, and that best managed by the general practitioner, has been described. He shows how plaster-of-Paris may be used in a variety of ways, and in many cases may take the place of expensive appliances. The book itself is a volume of 354 pages, with 177 illustrations, mostly original.

ADAMS.

LETTER, WORD AND MIND BLINDNESS. James Hinshelwood, M.A., M.D., F. F. P. S. Glas. London. H. K. Lewis, 136 Gower street, W. C., 1900.

Four of the five chapters of this little book by Hinshelwood appeared in the London *Lancet*, but the papers are well worthy of publication in book form. The author believes that past visual impressions are arranged in definite groups within the visual memory area, so that one or more of these groups may be destroyed without involvement of the others, and in this way letter, word or mind blindness is produced. For a similar reason the memory of figures or musical notes may be preserved while that of words is lost. Hinshelwood attributes much importance to the left angular gyrus, and in this he accepts Dejerine's views, but is in opposition to the teaching of some others. The perceptive center of visual impressions he says is in the neighborhood of the cuneus and calcarine fissure—this probably most will accept—but the angular gyrus and its neighborhood is a center of a higher nature, one of whose functions seems to be the storage of the visual memory of things and words. Is there anything more wonderful than the ability of nerve cells to store up impressions and to bring them again into consciousness when occasion demands? To have complete word blindness, according to Hinshelwood, the left angular and supramarginal gyri, or their connections with both right and left occipital lobes, must be destroyed. Hinshelwood thus accepts without question the left angular gyrus as the visual word center. He reports a number of interesting cases, none however with necropsy; one of the most important of these being a case of letter blindness without word blindness.

Anyone interested in sensory aphasia, especially that form dependent on injury of the visual word center, will find Hinshelwood's little book entertaining and instructive.

SPILLER.

TREATMENT OF PELVIC INFLAMMATION THROUGH THE VAGINA. By Wm. R. Pryor, M.D. W. B. Saunders, Philadelphia, 1899.

The treatment of pelvic inflammation through the vagina has been very well handled by Dr. Pryor. The "spirit of aggressive surgical interference" is predominant throughout this work, and the rules laid down governing this interference are very clear and concise. The author states his own ideas and methods of treatment in such a manner that the student has something tangible that he can seize upon and easily assimilate, and is not in danger of becoming confused by a number of conflicting theories. In fact, the complete absence of any theorizing is particularly commended.

He impresses upon the student the great benefits secured from good free drainage, not only from the uterus, but by opening the pelvic cavity and establishing free drainage through the cul-de-sac, by means of gauze packing. The incision at the same time allows the thorough exploration of the pelvis, and, if the condition found warrants it, the performance of a more radical operation.

His description of the successive steps of vaginal hysterectomy is especially good, and the cuts illustrating the different steps are all that could be desired. This book should be in the hands of every general practitioner and anyone making a special study of gynecology.

C. F. ADAMS.

JAHRESBERICHT ÜBER DIE LEISTUNGEN UND FORTSCHRITTE AUF DEM GEBIETE DER NEUROLOGIE UND PSYCHIATRIE. Herausgegeben von Dr. E. Flatau und Dr. L. Jacobsohn. Redigiert von Professor Dr. E. Mendel. II Jahrgang. Bericht über das Jahr 1898. S. Karger. Berlin, 1899.

The prompt appearance of the year book permits the reiteration of the praise accorded to it in our review of the first volume. For those who may not know of the enterprise it may be said that the editors have tried to collect the entire literature of neurology and psychiatry for 1898. They have classified it and have given abstracts of the more important books and papers which appeared during the year stated. The gathering of the titles of the numerous papers has been done with even greater comprehensiveness than for the first year, and there are abstracts of over a thousand more articles in this volume than there were for the literature of 1897. American and English literature has been very well represented; in fact, no literature has been neglected, and it is with especial satisfaction that we note the careful and extended résumés of articles published in languages which are not known to many students; such as from the Russian, Hungarian, Bohemian, Polish and Scandinavian. This present volume is better, if it were possible, than that of the past year and it is to be heartily commended. We repeat that it is indispensable for every working neurologist or alienist who desires to keep *au courant* with the literature of his subject.

JELLIFFE.

#### MISCELLANY.

The meeting of the American Medico-Psychological Association, to be held in Richmond, Va., has been postponed to May 22-25, 1900.

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THE  
Journal  
OF  
Nervous and Mental Disease.

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Original Articles.

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CONTRIBUTION TO THE STUDY OF THE PLANTAR REFLEX, BASED UPON SEVEN HUNDRED EXAMINATIONS MADE WITH SPECIAL REFERENCE TO THE BABINSKI PHENOMENON.\*

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Attention was first called by Babinski, in 1896, to the fact that in pyramidal tract disease stroking the sole of the foot often produces extension of the great toe, with or without extension and separation of the other toes. The status of this phenomenon as a practical aid in diagnosis has not yet been definitely determined. The diversity of opinion is shown by the conclusions of two recent investigators; Boeri, who represents the majority, states that it occurs in at least 76 per cent. of hemiplegics, old and recent, that it is a test of special value in the comatose stage of hemiplegia, and equally useful in distinguishing organic disease from functional disorder. Cohn, on the other hand, while allowing that extension is noted more often than flexion in disorder of the lateral columns *whether organic or functional*, adds that in no way can this phenomenon have any real pathognomonic value in diagnosis.

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\*Read at the twenty-sixth annual meeting of the American Neurological Association, May 1, 2 and 3, 1900.

In the hope of adding something to the knowledge of this subject, we have investigated 200 hemiplegics and diplegics, old and new; 30 cases of spinal disease involving the pyramidal tract, 70 infants, and 400 cases in which no recognized lesion of the pyramidal tract existed, the last group including 200 cases of nervous disease both organic and functional, 100 normal individuals, and 100 cases taken at random from hospital patients outside the neurological department. This study confirms the claim that the Babinski phenomenon furnishes a most important positive sign and definite aid in diagnosis. It would seem that this plantar reflex stands for something even aside from other symptoms, a distinction which we can hardly accord even to the knee-jerk, the importance of which, when taken as a link in the chain of symptoms, is paramount. Unlike the ankle-clonus it has no spurious analogue in functional affections, it is rarely difficult to elicit when present, it appears early in the disease, when every diagnostic sign is needed, and may be present during the later stages when other reflexes have disappeared on account of ankylosis or muscular atrophy.

Previous to the contributions of Babinski little attention was paid to the plantar reflex. It was taken as a matter of routine, like other skin reflexes, and the direction of the toe movement was not especially noted; it seems doubtful, however, if its presence, absence, and degree of activity are likely to prove often of great diagnostic value, as our collection of cases illustrates, but the demonstration of the Babinski phenomenon is of prime importance.

The first communication of Babinski<sup>1</sup> called attention to the fact that, in pyramidal disease, stimulation of the sole of the foot provoked extension of the toes, and particularly of the great toe, the other muscular contractions (flexion of thigh on pelvis, leg on thigh, foot on leg) persisting as in health. He named this reflex *le phénomène des orteils*. Two years later<sup>2</sup> he called attention to certain characteristics of the reflex movement, for example, that extension is slower than flexion, and that flexion is stronger when the inner half of the sole is excited, the converse being true of extension. He cited varieties

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<sup>1</sup>Babinski, Compt. rend., Soc. de Biol., Par., 1896.

<sup>2</sup>Babinski, Semaine med., Par., 1898, xvii, pp. 321-22.

of reflex partly physiological and partly pathological, noting, for example, that sometimes stroking one part of the foot would produce flexion, another part extension, the first reaction obtained in a given case being generally flexion; again, he sometimes noted extension of the two inner, with flexion of the outer, toes in response to a single stimulation. His observations have been confirmed by Van Gehuchten,<sup>3</sup> Glorieux,<sup>4</sup> Genault,<sup>5</sup> Létienne and Mirouche,<sup>6</sup> Buzzard,<sup>7</sup> Kalischer,<sup>8</sup> Cestan and Le Sourde,<sup>9</sup> Collier,<sup>10</sup> Boeri,<sup>11</sup> and Brissaud,<sup>12</sup> and in this country by Langdon.<sup>13</sup>

Its practical importance is denied by Schüler,<sup>14</sup> and by Cohn.<sup>15</sup> Giudiciandrea<sup>16</sup> has contributed to the uncertainty by stating that he has found extension in hysteria and in normal individuals.

Collier found in infants extension the earliest response; in pyramidal tract disease he found a similar reflex to that obtaining in infants, but more deliberate, extension of the great toe preceding other movements. He regarded this reflex as one of the first signs to appear, and the last to disappear, in temporary lesions, and stated that it might be the only unequivocal objective sign of pyramidal disease. He found it was the only reflex present in total transverse lesion of the cord. He regarded tetanus and large doses of strychnine the only additional possible sources of the extensor reflex.

<sup>3</sup>Van Gehuchten, *Jour. de Neur.*, April 5, June 20, July 5, 1898.

<sup>4</sup>Glorieux, *idem*, Dec. 5, 1898.

<sup>5</sup>Genault, *Th. de Paris*, 1898.

<sup>6</sup>Létienne and Mirouche, *Arch. gén. de méd.*, 1899, No. 2, p. 191.

<sup>7</sup>Buzzard, *Brit. Med. Jour.*, 1899, No. 2001, p. 1077.

<sup>8</sup>Kalischer, *Virch. Arch.*, 1899, Vol. 155.

<sup>9</sup>Cestan and Le Sourde (Cestan), *Th. de Paris*, 1899, and *Arch. de méd. de Toulouse*, April 5 and May 15, 1899; *Bull. de la Soc. Anat.*, 1898 (C. & Le S.; *Gazette des hôpitaux*, 1899, jeudi, 23 nos., Nr. 133, S. 1249.

<sup>10</sup>Collier, *Brain*, xxii, 71, No. 85, 1899.

<sup>11</sup>Boeri, *Riforma medica*, an. XV, vol. II, Nos. 71, 72, 73, pp. 843, 855, 867; June 26-28, 1899.

<sup>12</sup>Brissaud, *Gaz. hebdom. de méd. Chir.*, Paris, 1896, xliii, 253.

<sup>13</sup>Langdon, *Cincinnati Lancet Clinic*, February 17, 1900.

<sup>14</sup>Schüler, *Neur. Centralbl.*, 1899, Nr. 13, S. 585.

<sup>15</sup>Cohn, *Neur. Centralbl.*, 1899, Nr. 13, S. 580.

<sup>16</sup>Giudiciandrea, *Bull. Soc. Lancisiana*, 1899, fasc. I, p. 226.

The most complete corroboration of Babinski's claim has been recently furnished by Cestan and Le Sourde, who found the extensor reflex in 92 per cent. of the 68 hemiplegics examined, and in all the 35 cases of spinal disease implicating the pyramidal tract (except one case with neuritis also), in all the 6 cases of syringomyelia, and in all the 30 cases of infantile hemiplegia and diplegia.

These observers found the extensor reflex in infants from one month to one year of age, but never found it in normal adults or in those suffering from disease other than of the pyramidal tract.

Remak<sup>17</sup> found the Babinski phenomenon in the same patient (suffering from lesion at about the seventh dorsal roots), who presented the so-called femoral reflex described by him in 1893. In others words, while stroking the thigh produced flexion stroking the sole produced extension of the toes.

Schüler found flexion in 80 per cent. of normal men, extension (notably of the great toe) in 8 per cent., reflex wanting in 10 per cent., indeterminate in 2 per cent. In women 80 per cent. flexion, 10 per cent. wanting, 4 per cent. extension, 6 per cent. doubtful. In infants this observer found no reflex in 40 per cent., extension in 12 per cent., flexion in 30 per cent., variable in 18 per cent. In the 8 cases of pyramidal tract disease examined (amyotrophic lateral sclerosis, combined tabes, hemiplegia and monoplegia) the extension was pronounced.

Cohn found flexion in 60 per cent. of healthy persons; extension of the great toe, with either extension or flexion of the others, in 20 per cent.; no reflex in 10 per cent.; variable movements in 10 per cent. According to this observer the reflex is generally absent in infants, but when present it usually consists in extension of the great toe. He generally found extension in cases of recent apoplexy, and found it in two cases out of four of spinal spastic disease. In one case of hysterical hemiplegia he records extension on the paralyzed side.

Schäfer<sup>18</sup> has recently called attention to a phenomenon

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<sup>17</sup>Remak, *Neur. Centralbl.*, 1 January, 1900.

<sup>18</sup>Schäfer, *Neur. Centralbl.*, Nov. 1899.



in hemiplegics, which he names the antagonistic reflex, consisting in extension of the great toe produced by pinching the tendo Achillis. This reflex he finds very promptly within a day or two of the onset of hemiplegia. Since this communication Babinski has shown that pinching the skin alone over a considerable area, including that of the tendo Achillis, suffices to produce extension. We have observed the so-called antagonistic reflex frequently, but not constantly, and never in the absence of the Babinski phenomenon. This discovery, therefore, while of interest as demonstrating the variety of afferent tracts through which the Babinski reflex may be elicited, adds nothing of practical value to our knowledge, and the adoption of a special title for this method of stimulation only renders the nomenclature unnecessarily cumbersome. Schäfer's method is also objectionable on account of the painful degree of pressure usually requisite.

The object of our communication being rather to record clinical facts than to participate in pathological discussion, no reference is made to the anatomy or physiology of the reflex. We have also limited our observations in these records, though not in our practice, to the one set of muscles under discussion, namely, those moving the toes.

We shall first take up the reflex in normal individuals and those in which pyramidal disease was absent, we shall next consider the reflex in infants, and lastly that in pyramidal tract disease, cerebral and spinal.

The most satisfactory instrument in our experience for testing the plantar reflex is the orange stick not too sharply pointed. A deliberate and even stroke is made from the heel toward the toes, the outer and middle areas being specially tested to stimulate the reflex in the great toe. It is sometimes found in susceptible individuals that accompanying the point of the stick with the finger tip affords a better control and more uniform application, and in such individuals a slight stroke at the beginning is preferable, to avoid the semi-voluntary movement which masks the true reflex. We cannot help suspecting that this semi-voluntary movement, which may be either downwards or upwards, has been mistaken for the true reflex, in some instances at least, by those observers who have noted

extension in normal individuals. The true extensor reflex under consideration consists of a *deliberate, marked, extension of the great toe*, with or without extension and separation of the other toes. In some cases this movement is constant and uniform, however many times the sole is stroked; in other cases the reflex may tire, but whether persistent or non-persistent it is not to be mistaken for the variable and inconsequent movements sometimes observed in sensitive persons. *Such a reflex we have never found in normal individuals.*

*The normal plantar reflex.*—Opportunity to study the normal reflex under the most favorable conditions was kindly furnished us by Miss A. M. Homans, Director of the Boston Normal School of Gymnastics. Two classes, mainly young women, 20 to 25 years of age, were examined; 27 were examined in one class before gymnasium work, and 28 in the other after an hour's continuous exercise.

The results in the two groups were so similar as to lead to the conclusion that moderate fatigue has no effect on this reflex. In 40 (20 in each class) of the 55 cases the typical normal reflex was present, namely, flexion of all or some toes, generally the outer, the others remaining quiet.<sup>19</sup>

In 5 cases out of the 55 (slightly under 10 per cent.) no movement was elicited, this group comprised 3 from one class and 2 from the other.

The instructive group in which a typical normal reflex (flexion of all or of some toes) was present in one foot while no response occurred in the other contained 10 cases, *i. e.*, almost 20 per cent. Of these cases 5 were found in the class examined before, and 5 in the class examined after exercise.

Other normal individuals were tested to bring the number to 100. Both sexes and all ages over two years were included.

The percentages were as follows: All, or some, toes (generally outer) of both feet flexed, 75 per cent.; no movement, 10 per cent.; flexion on one side, no movement on the other, 15 per cent.

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<sup>19</sup>In this group, as a rule, the two sides correspond; for instance, in one case the outer three toes only of both feet were flexed, in another individual the reflex was limited in both feet to the great and second toes.

A second series of 100 cases, taken at random from the hospital, with other than nervous disease, gave approximately the same result, differing only in that fewer cases showed absence of reflex on one side and persistence on the other. These percentages were as follows: Flexion of all or some toes (generally outer) on both sides, 87 per cent.; absence of movement, 9 per cent.; flexion on one side and absence of movement on the other, 4 per cent.

The conclusion to be drawn from these cases is that the usual normal reflex consists of flexion of all or some (generally outer) toes on both feet—that in perhaps 10 per cent. of cases no movement occurs, and that in at least this proportion flexion occurs on one side and no movement on the other.

The fact that the plantar reflex is wanting in an appreciable percentage of normal individuals has been already noted by various observers; such absence has probably rarely led to a mistake in diagnosis among those at all familiar with nervous disease. It has not been, we believe, hitherto recorded, though doubtless often observed, that the reflex is not infrequently in health present on one side and absent on the other, and we feel sure that experienced clinicians have mistaken such a difference for indication of disease.

To determine the *plantar reflex in disturbance of the nervous system with no demonstrable lesion of the pyramidal tract*, 200 cases were examined, this list including such diseases, organic and functional, of the nervous system as were found in the neurological clinic at the Massachusetts General Hospital or in outside practice. These diseases included atheroma, alcoholism, acromegaly, bulbar paralysis, basal disease, concussion, cerebro-spinal meningitis, chorea, dementia, epilepsy, general paralysis, Huntington's chorea, hemorrhage in cord (lumbar), hemiplegia with recovery, head injury, hypochondria, hysteria, joint atrophy, monoplegia, paresthetic meralgia, melancholia, neuritis, neurasthenia, progressive muscular atrophy, post-diphtheritic paralysis, poliomyelitis, paralysis agitans, retarded development, Raynaud's disease, rachitis, sensory hemiplegia, senility, sciatica, specific disease of cord, tabes, tumor of the brain, and uremia.

In  $65\frac{1}{2}$  per cent. of these cases flexion of all or some (generally outer) toes occurred, in  $16\frac{1}{2}$  per cent. there was absence

of movement on both sides, in 9 per cent. flexion occurred in one foot, no movement in the other; in 7 per cent. quick and variable movements appeared. The Babinski phenomenon occurred in 5 cases (2 per cent.). In these 5 cases (general paralysis, meningitis, hydrocephalus, uremia, alcoholism) implication of the pyramidal tract could not be excluded, and in view of previous study the suggestion naturally presents itself that this very sign may establish its existence. Further reference will be made to these cases.

Comparison of these percentages with those in normal individuals shows a striking similarity, and demonstrates the persistence of the normal reflex in disease.

It is of interest to note in passing that in 7 of the 18 epileptics included in this group the reflex was absent in one or both feet, that of the 15 tabetics 5, and of the 16 cases of hysteria 10, showed a like absence of reflex. This percentage of absence is far beyond the normal, though the cases are too few to add materially to statistics bearing upon these conditions.

Leaving these 49 cases out of consideration then, we had left 151 cases of various diseases of the nervous system with no more than the normal percentage of absent plantar reflex.

Opportunity to study the *infantile plantar reflex* was kindly furnished us by Dr. W. L. Richardson, and by Dr. Higgins, at Boston Lying-in Hospital, and by Drs. Morse and Bartol at the West End Nursery.

Our results coincide here rather with the observations of Cohn and Schüler than with those of Collier. In the first place we find it extremely difficult, and often impossible, to determine the nature, or even the existence, as regards the toes, of a definite plantar reflex in early infancy. In young infants automatic movements of the toes are almost constant, varying from extreme extension and separation to the opposite position, whether the sole is stroked or not; even after allowing for this source of error the results are baffling, as was illustrated by an experiment of Dr. Richardson.

After we had examined a number of infants with varying results, he announced that the last case on the list had been twice tested, having been replaced in the line by the nurse at

his suggestion. Comparison of the results showed that where-as in the second test the observation was "toes generally up," the note in the first was "toes generally down." Four other infants were then tested, each twice, without the examiner knowing the order of arrangement; in only two of these four cases were the second observations like the first. In marked contrast to these results dorsi-flexion of the *foot* was constant. Over forty infants were tested, varying from a few hours to two weeks old, with similar results, the upward movement perhaps predominating, but not with sufficient constancy to establish a rule. In our opinion, therefore, the Babinski phenomenon is not constant in early infancy; in fact, we question the existence of a characteristic toe reflex at this period.

It would appear that before the pyramidal tract is developed the reflex movements of the toes, if indeed such variable movements deserve the name reflex, are merely the generalized motions characteristic of early infancy, responsive, probably, to afferent impulses, but in no way to be compared either to the normal plantar reflex or to the Babinski phenomenon.

At just what age the reflex is established we are uncertain, but our study of thirty older infants tends to show that it is not developed until after the first year. This point deserves further study.

We come now to the consideration of the *plantar reflex in pyramidal tract disease*. The opportunity to study the most interesting and instructive series of cases we have to communicate was furnished us through the courtesy of Dr. Fernald, Superintendent of the Massachusetts School for Feeble Minded. This series comprised 33 cases, of which 24 were diplegic and 9 hemiplegic.

Of the 9 hemiplegics, in two cases extension of all toes occurred on the paralyzed side with normal flexion on the other side; in two cases extension of the great toe occurred on the paralyzed side, while on the healthy side flexion obtained in one of these cases, no movement in the other; in the fifth and sixth cases marked extension occurred on the paralyzed, with slight extension on the healthy side; in a seventh case (a patient 21 years of age, hemiplegic since birth) the plantar reflex

was wanting on the paralyzed side, flexion occurring on the other; in an eighth case (a woman 45 years of age, hemiplegic since birth) the reflex was wanting on the paralyzed side, slight extension occurring on the other; in the ninth case typical extension was exhibited on both sides.

The most striking exhibit was that of the diplegics. Of the 24 cases, in 15 the typical extensor reflex obtained in both feet; in 2 cases the great toe was extended, the others making no movement; in 1 case all toes were extended and separated in one foot, in the other foot separated without extension; in 1 case extension of all toes occurred on one side, no reflex on the other; in one case extension of all toes occurred on one side, while on the other side extension of the great toe only was present, the others being flexed; in 3 cases (aged 12, 17, and 24 years respectively) plantar reflex was absent as regarded the toes, though dorsal flexion of the tarsus was present; in one case it was impossible to take the reflex on account of violent choreic movements.

We would call attention to one or two points of interest in certain of the cases above summarized; in one of the 15 cases with typical extensor reflex stroking of the right foot produced a reflex on both sides (crossed reflex); in 2 of these same 15 cases the feet were in a state of contracture with marked extension of all toes, the great toe especially being in a state of hyperextension. These were both old cases, one 25 and the other 45 years of age, both paralyzed since birth; stroking the sole in one of these cases produced a very slight increase in the extension; in the other, on overcoming the extension by gentle pressure of the hand, stroking the sole brought the toe forcibly up against the hand. In the three cases with absence of movement there was extreme rigidity. In connection with these three cases it is interesting to note that in two hemiplegics reflex movement was wanting on the paralyzed side also. In these cases the paralysis was of long standing (21 and 45 years) and rigidity was marked. It is not at all impossible therefore that early in life extension was present on the paralyzed side.

In this series no case of pyramidal tract disease showed flexion of all toes of both feet. Extension in more or less com-

plete form was present except in an occasional case in which no movement of the toes occurred. These results certainly go far toward establishing the fact that extensor reflex, when present, is pathognomonic of pyramidal disease; that absence of movement, especially in old cases, does not preclude such disease, while flexion of the toes is strongly indicative of pyramidal tract integrity.

It is of interest in this connection to observe the variation in knee-jerk and ankle-clonus as contrasted with the constancy of the plantar reflex, for while the knee-jerk was exaggerated in many cases, it was, in a considerable number, absent on one side or both, ankylosis of the knee-joint and muscular atrophy being present; ankle-clonus was generally wanting, the tarsus having become rigid.

Another interesting group of cases, at the Long Island Hospital, was furnished us through the kindness of Dr. Taylor, the visiting physician. In this group were 13 hemiplegics and 2 spinal cases. The duration of paralysis ranged from 3 weeks to 23 years, the ages from 26 to 78 years.

In the 13 hemiplegics extension of all toes on the paralyzed side with flexion or indeterminate action on the healthy side, occurred in 5; extension of the great toe on the paralyzed side with flexion or absence on the healthy side, in 3; extension of the great toe on the paralyzed side and the same in less degree on the healthy side, in 3; slight downward movement of all toes on both sides in 2 (6 months' and 4 months' standing).

In one of the spinal cases in which the great toe on the paralyzed side was extended the others were flexed when the inner, and extended when the outer, half of the sole was stroked. In the other, a congenital case of 23 years, with typical contracture and absence of knee-jerk, when the outer edge of the sole was stroked on the paralyzed side all toes were deliberately extended and separated, but when the inner side was stroked the opposite movement took place. This phenomenon was constant. On the healthy side all the toes were flexed, the great toe either extending slightly or remaining immobile.

A third group of hemiplegics was studied at the Tewksbury State Almshouse Hospital through the courtesy of Dr. Nichols. This group comprised 35 cases, varying in age from 24

to 80 years, in duration from one week to 15 years. In some cases the motion of the affected members had been practically restored.

Extension of all toes on the paralyzed side with extension or flexion of some or all on the healthy side was observed in eight cases; extension of the great toe on the paralyzed side, with flexion or absence of movement on the healthy side, existed in ten cases; extension of the great toe on the paralyzed, and the same in less degree on the healthy side, existed in six cases (in the last two classes the second was extended with the great toe in a large proportion of the cases, the outer toes being more often flexed).

The following variations of plantar reflex were noted in individual cases: no response on the paralyzed side, but flexion on the healthy side; straightening of outer toes with flexion and then extension of great toe on the paralyzed, with flexion on the healthy side; four outer flexed, great toe motionless on both sides; great toe stationary and others slightly extended on the paralyzed side, with great and second on the healthy side flexed and others immobile; great and second toes stationary, others straightening on the paralyzed side. Three cases only (1, 2, and 8 years' duration) exhibited flexion of all toes of both feet.

The preceding groups of cases are mentioned in detail as presenting fairly characteristic variations of plantar reflex in disease of the pyramidal tract.

We have examined in addition a sufficient number of hemiplegics and diplegics to bring the number to 200. In making this collection we have availed ourselves of the courtesy of the following superintendents of hospitals for the insane: Dr. Harrington, Danvers; Dr. Brown, Taunton; Dr. French, Medfield; Dr. Drew, Bridgewater; Dr. Quimby, Worcester; Dr. Page, Middletown, Conn.; and Dr. Flood, Hospital for Epileptics, Munson. We have also examined cases kindly placed at our disposal by the visiting physicians of the Massachusetts General Hospital and the Boston City Hospital, besides recording all cases coming under our own observation in the Neurological Department of the Massachusetts General Hospital and in outside practice. We have also to thank Dr. Courtney



for the privilege of examining cases at the Boston Home for Incurables, and Dr. Thayer of West Newton for furnishing material from his practice.

Out of the 200 cases examined, 140 (70 per cent.) showed the Babinski phenomenon. The varieties of response in both the Babinski and non-Babinski cases were practically identical with those enumerated in the three groups detailed above.

*The Babinski phenomenon in spinal disease involving the pyramidal tract.*—We have examined 30 patients of this class with the following result: In lateral sclerosis we found 2 cases with the Babinski phenomenon out of 4; in syringomyelia, 1 out of 2; in ataxic paraplegia, 2 out of 5; in combined system disease, 2 out of 3; in myelitis, 5 out of 6; in trauma, 3 out of 6. In 2 cases of spinal spastic paralysis of unknown pathology, and in 1 case of hemorrhage into the cervical cord, the Babinski phenomenon was present; in 1 case of disseminated sclerosis, involving the pyramidal tract, it was absent.

The Babinski phenomenon obtained, therefore, in 60 per cent. of these cases.

*The persistence of the Babinski reflex at various periods and under varying conditions* was illustrated by the fact that clonus was absent at the time of our examination in 40 of the cerebral cases, and in 7 of the spinal (18) cases, showing the Babinski phenomenon. The exaggerated knee-jerk was more constant than the clonus; still, even this sign failed in 17 of the cerebral and in 5 of the spinal cases. These facts serve to emphasize the value of the Babinski sign as a practical aid in diagnosis.

*Rare cases showing the Babinski phenomenon, but with no demonstrable lesion of the pyramidal tract.*—The foregoing tabulation of cases would indicate that the absence of the Babinski phenomenon does not throw out pyramidal disease, but that its presence establishes this lesion with almost absolute certainty. We could say with *absolute certainty* were it not for an occasional baffling case in which extension occurs without recognizable lesion of the pyramidal tract. In our investigation five such cases have come to light, and Dr. Vickery has given us

the record, in addition, of its discovery in a case of profound opium poisoning.

The first case was that of a child of 3 years, with meningitis, under the care of Dr. Vickery. The principal symptoms were unconsciousness, strabismus, opisthotonos, restlessness and convulsions. There was no König symptom. The knee-jerk was rather active. The plantar reflex consisted of decided extension of both great toes with flexion of the other toes on both feet.

The second case was that of a boy two years of age, a patient of Dr. F. L. Jack's, at the Eye and Ear Infirmary. This was a rachitic, hydrocephalic, poorly nourished infant with large abdomen, and pronounced rosary. There was a slight knee-jerk. Stroking the sole produced strong extension of all toes on both feet.

The third case was that of a general paralytic, a man 49 years old, with general weakness, but no definite hemiplegia. The knee-jerk was normal; the plantar reflex consisted of decided extension of the great toe on the right, the others spreading slightly; the great toe of the left foot straightened, but was not extended, the others having been slightly flexed.

The fourth case was that of an alcoholic patient with loss of consciousness lasting 24 hours, during which time a marked Babinski phenomenon was present and constant on both sides. (This case is mentioned in more detail further on.)

The fifth case was one of acute uremic poisoning in the service of Dr. Cutler at the Massachusetts General Hospital. The patient, a man of 60, was brought in unconscious, with exaggerated knee-jerks, ankle-clonus, and marked Babinski phenomenon on both sides. A series of convulsions ensued during which the plantar reflex disappeared entirely, to be replaced by the Babinski phenomenon in the intervals. The following day the patient was conscious, complaining only of malaise and moderate headache. The knee-jerks were not exaggerated, there was no trace of clonus, and the plantar reflex consisted of prompt flexion of all toes.<sup>20</sup>

It will be noted that even in these cases there was reason

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<sup>20</sup>The disappearance of the Babinski phenomenon *during* convulsions has been noted by Keniston, as well as its appearance.

strongly to suspect damage to the pyramidal tract, direct or indirect, as by edema, and it seems not improbable that more complete knowledge may lead us to make such a diagnosis from this sign alone; but to draw this conclusion now would perhaps be going too far.

In illustration of the *aid furnished in diagnosis by this phenomenon*, we cite the following cases selected to demonstrate its significance in both cerebral and spinal lesions:

The first two cases (both middle-aged men) presented, on the initial visit, great clinical similarity, namely, headache, malaise, confusion of ideas, mental apathy preceded by restlessness, slow pulse, slight rise in temperature, and moderate aphasia without paralysis. The tendon reflexes were in neither case remarkable. The plantar reflex in one case was normal, while in the other the Babinski phenomenon was noted in the right foot. Both cases died within the week, in the first case without paralysis, in the second case with marked right-sided hemiplegia. Autopsy in the first case demonstrated internal hydrocephalus; no autopsy was obtained in the second, but the sequence of symptoms (complete right-sided hemiplegia preceded by a convulsion) established pyramidal tract disease, probably hemorrhage.

Two spinal cases, in which the Babinski phenomenon aided greatly in diagnosis, showed identical symptoms and probably resulted from identical lesions. Both these patients were women over 50 years of age, anemic, and so markedly neurasthenic that for some time organic disease was unsuspected. At the time they came under observation they were confined to bed with relaxed paralysis and incontinence of urine and feces; muscle sense was lost in the feet, and there were areas of dissociation on the lower extremities, but no marked loss of tactile sense. The knee-jerks were wanting in one case and were much diminished in the other. The Babinski phenomenon was present in both cases, and helped confirm the diagnosis, namely, Putnam's combined system disease in its terminal stage of general softening. It is not improbable that the Babinski phenomenon was present early in the course of these cases, in which event it would have established the diagnosis

of organic disease at a time when all other evidence pointed in the opposite direction. It would seem, then, that this sign may not only aid in early diagnosis of organic lesion, but by its persistence in advanced amyotrophic poliomyelitis, combined system disease, and similar conditions, establish the pathology of which every other definite evidence has disappeared through involvement of the lower neurons in the destructive process.

An excellent illustration of the *permanence of the Babinski sign* through the various stages of pyramidal disease was furnished by a case of traumatic hemiplegia in the service of Dr. Beach at the Massachusetts General Hospital. The skull had been crushed by a falling derrick. Dr. Scudder removed a large plate of bone over the motor area on the right side, together with considerable lacerated brain tissue. Hemiplegia was present from the first, with relaxed paralysis and absence of knee-jerk in the earlier stages, replaced gradually by spastic condition. When first seen by us, shortly after operation, the Babinski sign was already present, though the knee-jerk was absent. When seen at the end of several months, after establishment of exaggerated knee-jerk and clonus, the Babinski sign was found to have retained unchanged its original character, namely, deliberate extension of the toes on the paralyzed side, most marked in the great toe.

The early establishment of this sign was shown by a case seen with Dr. Baxter, of Hyde Park. Crossed right hemiplegia appeared in the early morning, and when the patient was visited at 10 o'clock the Babinski sign was already established. Again, in a case in Dr. Cutler's service, the Babinski phenomenon was marked five hours after the apoplectic shock.

That the early appearance of this symptom is not invariable is shown by the following case seen in consultation with Drs. Beach and Mumford:

This was the case of a man of 60 presenting a typical history of hemorrhage from one of the perforating branches of the posterior cerebral artery, probably the external optic. The order of symptoms was right hemianopsia, numbness of the right side, restlessness, depression, followed gradually by elevation of temperature, deepening stupor, and right-sided motor hemi-

plegia. Death took place two weeks from the date of onset. During the first two days the plantar reflex was absent on the right side, normal flexion occurring on the left. The onset of motor paralysis was accompanied by deliberate straightening of the outer toes on the right, the great toe giving no response; this reflex remained unchanged till the day of his death. While this case may be regarded perhaps as showing an approach to the Babinski phenomenon, it cannot be cited as a typical case, but offers rather an exception to the rule, provided there was a destructive lesion of the anterior capsule. Unfortunately no autopsy was made.

The suggestion naturally presents itself that the motor paralysis was due to pressure upon, rather than invasion of, the anterior portion of the internal capsule. Whether this distinction has a bearing on the nature of the plantar reflex we are not yet in position to determine.

The following acute case (already alluded to under alcoholism) shows the importance of examining for *the Babinski sign in the unconscious state*, in which it seems likely to prove of the greatest value in diagnosis, though our material is still too scant to permit the formulation of absolute rules.

The history of this case was as follows: W. M., a pronounced alcoholic, was drawing a loaded truck down an inclined plane, when he was thrown forward, the truck striking him and fracturing his right thigh. There was no loss of consciousness. He was admitted to the Massachusetts General Hospital in the service of Dr. Homans, and was doing well up to the afternoon of the third day, when he gradually became unconscious, the temperature rising to 102 degrees. There were no local spasms, and there was no restlessness. When seen in consultation with Dr. Homans the patient was completely unconscious, the pupils contracted and irresponsive to light. There was no strabismus. The extremities, though generally quiet, were moved occasionally—that is, there was no hemiplegia. The patient gesticulated at times with his left hand as if in delirium. Both arms at times offered resistance to passive motion, though they were generally lax. The knee-jerk was absent on the left; it could not be tested on the right on account of the fracture apparatus. *The Babinski reflex in*

*marked degree was present in both feet.* The next day consciousness had returned, there was paresis of the left external rectus, the left pupil was slightly larger than the right, both reacting to light. Movements were present, though sluggish on both sides. *The knee-jerk had returned and the Babinski sign had disappeared*, all toes being flexed on the right, and on the left all but the great toe, which showed no movement.

The next day the patient became restless, and on the following day was violent. On February 12 the pulse became weak and rapid, the temperature and respiration steadily rose, coma supervened, and death took place at 9.30 P. M., the temperature having reached 108.5 degrees. No paralysis appeared during this time.

Autopsy was not allowed, so that we are in doubt as to the exact pathology. This was peculiarly unfortunate, as the case seemed likely to add materially to our knowledge of the diagnostic value of this reflex. The bilateral appearance of the phenomenon without paralysis, and its disappearance in one day, certainly shows that serious lesion of the pyramidal tract was not present. The case tends to controvert the theory towards which our observations were tending, namely, that mere pressure, whether cortical or capsular, is unable to produce the Babinski reflex. The probable diagnosis in this case was alcoholism with bilateral cerebral edema, possibly accompanied by hemorrhage, but not by definite lesion of the pyramidal tract.

There is perhaps some analogy between the cerebral conditions in this case and those present during, and immediately succeeding, certain epileptic convulsions exhibiting the Babinski phenomenon. Recent observations on this point have been made but not yet published by Dr. Keniston, of Middletown.

That deep narcosis, as such, does not, necessarily at least, produce the Babinski phenomenon, is shown by the fact that we have invariably found *absence* of plantar reflex in patients completely etherized.

#### *Conclusions.*

1. In health either of the following conditions may appear

on taking the plantar reflex: (a) flexion of all toes; (b) flexion of some (generally outer) toes; (c) flexion of all toes on one side, and of some (generally outer) on the other; (d) entire absence of movement on both sides (about 10 per cent.); (e) flexion of all or of some (generally outer) toes on one side, with absence of movement on the other (at least 10 per cent.); (f) occasionally (in sensitive individuals) quick, semi-voluntary, indeterminate, movements, sometimes of flexion, sometimes of extension.

2. In early infancy no constant or characteristic movement of the toes appears, though extension is rather more frequent than flexion.

3. The Babinski reflex obtains in about 70 per cent. of hemiplegics and diplegics, and in approximately the same percentage of cases with disease involving the pyramidal tract in the spinal cord.

4. The Babinski reflex (*deliberate* and *constant* extension of the great toe, with or without extension and separation of other toes) is never present in health, and our observations lead us to doubt its existence in either functional or organic nervous or other disease not implicating the pyramidal tract.

5. This reflex is often the earliest to appear in pyramidal tract disease, *e. g.*, at the onset of a hemiplegic attack before the establishment of the exaggerated knee-jerk and ankle-clonus; it may persist during a period when other reflexes are absent, *e. g.*, when knee-jerk and ankle-clonus are wanting on account of ankylosis, contracture and muscular wasting, as in long standing diplegia, or of degenerative sequences as in combined system disease. This reflex furnishes, therefore, a most important practical aid in diagnosis.

6. This reflex very exceptionally appears in cases not conforming to recognized types of pyramidal disease (meningitis, hydrocephalus, poisoning, as by alcohol or uremia). These instances are too few materially to impair the diagnostic value of the phenomenon; in fact, the Babinski reflex alone should here rather lead us to suspect pyramidal involvement as by edema or indirect pressure.

## TWO CASES OF PRIMARY NEUROTIC ATROPHY BEARING A RESEMBLANCE TO MULTIPLE NEURITIS.\*

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The following cases are worthy of record because of the rarity of the affection, and because of the resemblance they both bear in their present condition to multiple neuritis:

CASE I.—M. McM., female, white, married, aged 47, birth-place Philadelphia, admitted to the nervous wards of the Philadelphia Hospital May 9, 1899.

Family History.—Mother died at fifty years of age of



Fig. 1. Hands in Case I., showing position of fingers in extension and interosseous atrophy.

dropsy. She has not heard of her father for many years. Nothing known of brothers and sisters.

Personal History.—Had smallpox, measles, whooping-cough and mumps when a child. Once had an abscess of the knee. Denies venereal history and alcoholism. Has had two

\*These cases were presented before the Philadelphia Neurological Society, Jan. 22, 1900. See p. 287.



children, both now dead; one of cholera infantum and one of accident. Had no miscarriages. Still menstruates regularly.

**Present Illness.**—About five years ago she noticed that her feet and legs were becoming weak. After awhile had to walk with a cane. The trouble slowly became more marked and two years later she noticed a change in her hands; she could not use them as well as before. She noticed that her toes dragged in walking and she found difficulty in going upstairs. At this time pain, quite severe, was present on the inner side of both legs below the knees; at times it was sharp. One morning she noticed that she could not use her right arm. It was very weak for about six weeks and then gradually she regained



Fig. 2. Feet in Case I., showing foot-drop.

control over it. Soon noticed also that the muscles of both hands were wasting, especially between the thumbs and index fingers. This wasting has continued.

**Status Præsens.**—Is a fairly well-built woman. In both hands there is marked wasting of the thenar and hypothenar eminences and of the interosseous muscles. The lower third of both forearms is also somewhat wasted. There is no wasting in the upper arm or shoulder. The grip is weak in both hands. In extension the fingers assume the position of

interosseous palsy; there is over-extension of the proximal phalanx and flexion of the other phalanges. The palsy and wasting of muscles is not limited to the ulnar supply, but is also marked, as has been stated, in the median and radial distributions. Pronation and supination are normal. Flexion and extension of the forearm upon the arm are also normal.

The muscles of the calves are somewhat flattened. All of the muscles of the legs are weak and there is a decided foot-drop upon either side. The patient walks with a typical step-page gait. The thighs are flabby. Both knee-jerks are absent.

There is decided tactile loss over both forearms, but this loss is merely a hypesthesia. There is no loss to thermal or pain sense.

There is hypesthesia of both legs, more marked over left knee and right lower leg. The pain sense is somewhat diminished, and there is also some blunting of the temperature sense. The responses over the thigh are prompt, except over upper outer aspect, where there is some hypesthesia.

There is no sensory loss over the trunk. The plantar reflex consists of flexion of leg at knee with a tendency to extend the feet. No movement of the toes is elicited. A wrist-drop is not present, although there is some weakness of the extensor muscles of the forearms.

Examination of the eyes by Dr. de Schweinitz revealed the following: Nebulous cornea from attack of smallpox. Pupil dilated. Dense haze through cornea prevents study of optic nerves. Apparently neither gross atrophy nor neuritis. Width of palpebral fissure is about three-eighths of an inch, slightly wider on the right side. This apparent drop is due to the former keratitis and probable former granular lids. It is not ptosis, the movements of the lids being perfect.

CASE II.—S. T., white, male, aged 37, born in Philadelphia, occupation bartender, single. Admitted to the wards of the Philadelphia Hospital July 27, 1899.

Family History.—Father died of congestion of the brain. Mother died of kidney disease. One brother died early in life, cause unknown. Another brother, four years older, suffers from an affection of the arms and legs very much like that of the patient.

Personal History.—No illness of moment in childhood save whooping-cough. In boyhood and early adult life was perfectly well. Was a bartender and drank moderately. Subsequently worked as fireman and was exposed to great heat; never, however, suffered from heat stroke. Never had anything to do with paints and never suffered from lead poisoning.

Several years ago he began to have pain in both thighs

and subsequently in the legs and feet. The pain was at times shooting, at times dull, never burning. It would last for an hour or two, disappear and then return after several hours had elapsed. For three or four months he has had pains in the arms, more especially on motion; he would "catch" his muscles.

Two years ago he noticed that the ankle of the right foot was becoming weak and tended to turn. Six months later he noticed the same thing in the left foot. He found that he had



Fig. 3. Hands of Case II., showing position of fingers in extension.

now to raise his feet higher from the ground than usual so as not to drag his toes. He had no other symptoms. Had perfect control over the bladder and bowel.

Status Præsens.—Sway slightly increased but not ataxic. Stands with difficulty on the right leg alone and with greater difficulty on the left. Slight intention tremor of both hands. Knee-jerks exaggerated. No ankle-clonus. Face, back, chest and abdomen present no abnormalities. Both hands are cold, damp and livid. Marked interosseous wasting, most pronounced in the ulnar distribution. In the right hand the fingers are irregularly extended. The thumb is extended normally. In the forefinger there is slight retroflexion at the metacarpal articulation, while the middle and ring fingers are retroflexed at the proximal and middle articulations. The

little finger assumes position of ulnar palsy though not typically. It is retroflexed at the metacarpal articulation and flexed at the phalangeal articulations. The palm is hollowed out because of wasting of the lumbricales. There is some flattening of the thenar and hypothenar eminences. In all the fingers the distal phalanx is slightly flexed. In the left hand all of the fingers present slight retroflexion at the proximal phalangeal



Fig. 4. Feet and legs of Case II., showing foot-drop, position of toes and exaggeration of plantar arch.

joint and flexion at the distal phalangeal joints. The thumb is held in a normal position. The palm is slightly hollowed; less decidedly than the right. Both thenar and hypothenar eminences show slight wasting; the grip is weak in both hands.

All of the cutaneous senses are preserved in all portions of arms, trunk and head.

The thighs are normal in size. Both legs are wasted, the

right more so than the left. In the right leg there is marked wasting of the tibialis anticus and entire peroneal group. The gastrocnemius is also wasted but less markedly. There is complete foot-drop—patient utterly unable to flex the foot on the leg. The plantar arch is very greatly increased. The toes are over-extended on the metatarsal joints and sharply flexed at the phalangeal joints. This is typically seen in the great toe. The normal curves of the foot are greatly exaggerated. The patient is unable to extend the toes; he can, however, flex them. No impairment of the muscles of the thighs. No movement of the toes on irritating the plantar surface. All forms of sensation are preserved. In the leg there is marked wasting in the peroneal group, though not as pronounced as in the right leg. Wasting in gastrocnemius less marked than right. There is also complete foot-drop, and, as in the right foot, there is an exaggeration of the normal curves of the foot.

The patient walks with a steppage gait, the feet being raised somewhat higher than normally so as to clear the ground with the toes.

Examination of the eyes was entirely negative.

These cases are exceedingly interesting because of the superficial resemblance which both bear to multiple neuritis. The history and general symptoms, however, leave no doubt as to their true nature.

It will be interesting to briefly summarize the post-mortem findings in this, as yet, obscure affection. Siemerling<sup>1</sup> has reviewed the various results of the limited number of autopsies that have thus far been held.

In Virchow's case there was degeneration of the columns of Goll, of the nerves and muscles and also atrophy of the anterior cornua.

In Dubreuilh's case there was slight increase of the glia in the columns of Goll, slight increase of staining in the pyramidal columns and pronounced changes in the peripheral nerves growing less from the periphery toward the centers. There was also atrophy of muscles.

In the case of Dejerine-Sottas there were changes in the posterior columns; sclerosis of the posterior roots in the lumbar region and sclerosis of the columns of Goll posteriorly in the cervical region. There was also interstitial hypertrophic

<sup>1</sup> E. Siemerling, *Archiv für Psychiatrie und Nervenkrankheiten*, Volume XXXI, 1898-1899, Heft. 1 and 2, page 105.

neuritis extending as far as the cord and involving the roots and spinal ganglia.

In Marinesco's case there was degeneration of the posterior columns, of the posterior horns, of the posterior roots, atrophy of the cells of the anterior horns, atrophy of the muscles and degeneration of the peripheral nerves with hypertrophy of the interstitial connective tissue.

In Gombault and Mallet's case there was sclerosis of the posterior columns and posterior horns, atrophy of the anterior horns, hypertrophic sclerosis of the roots and peripheral nerves, atrophy of the muscles.

In Siemerling's case there was degeneration of the posterior columns, most marked in the lower dorsal cord. In the upper dorsal and cervical cords there was almost complete degeneration of the lateral columns and in part of the antero-lateral bundle, most pronounced in the lower dorsal and lumbar cord. In addition, Siemerling found diminution of the cells in the anterior horns and in Clarke's columns, degeneration and diminution of the fibers of the anterior roots, destruction of cells in the spinal ganglia, degeneration of the peripheral nerves and marked atrophy of muscles.

The changes which appear to be common to all cases are a degeneration of the peripheral nerves and of the posterior columns together with atrophy of muscles. The changes appear to be most marked in the peripheral nerves and become less pronounced in passing from the periphery to the cord.

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- 57 NEUERE UNTERSUCHUNGEN ÜBER DIE PSYCHISCHEN WIRKUNGEN DES ALKOHOLS. (Newer Investigations on the Psychical Effects of Alcohol). Kraepelin (Münchener medicinische Wochenschrift, 1899, No. 42, S. 1365).

Reviewing the results of other experimenters and comparing them with his own, the author feels justified in drawing the following conclusions:

The grasping of external impressions and the linking of concepts suffer great deterioration through the use of alcohol. The production of muscular movements is temporarily made easier, but the power is lessened, and the more so the more the muscles come into action, while fatigue occurs more quickly. The action of one large dose of alcohol may be prolonged for 24 or 48 hours, while its continued use causes permanent diminution of capacity. ALLEN.

## A DIGEST OF RECENT WORK ON EPILEPSY.

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I propose herein to give a digest of the work done upon epilepsy in the two years from and including 1898 to the present month. In arranging a review of the literature upon epilepsy of a little more than two years, consisting of many hundreds of separate articles, I was surprised to find so little devoted to elucidating the problem of the hereditary or acquired instability of the cerebral cortex, the essential basis of most of the epilepsies. This is the more striking as it is steadily becoming more apparent to neurologists that the predisposition plays the most important rôle. It is generally conceded to-day that the hereditary element, which largely composes the predisposition, is found in 85 per cent. of all cases (Gowers).

### ETIOLOGY OF EPILEPSY.

Since the discovery of the cortical motor area and the pyramidal tracts it has been generally admitted that without the cortex there can be no epilepsy, and the impulses from the cortex must travel to the peripheral nerves along the pyramidal paths; but beyond this nearly everything is still in dispute, and the investigations of different workers are so variant as to make it impossible to harmonize their conflicting views and researches.

In a study upon the motor paths in epileptic attacks, Bischoff finds that cutting the pyramidal paths at any level renders the production of muscular spasm by the faradic current more difficult; isolated muscles may be made to contract in contra-lateral extremities. Despite complete division of the paths, unilateral or bilateral, at any level, persistent faradic irritation of the cortical motor area may lead to genuine epileptic convulsions. Faradization of the pyramidal tract itself produces tonic muscular contractions in the contra-lateral extremities. He found that isolated injury to the tegmentum seems to have no influence upon the production of single spasms in the contra-lateral extremities. If the tegmentum and pyramidal paths were both divided on one side and the

homo-lateral hemisphere were irritated, the convulsions were weaker on the contra-lateral side than they were on the homo-lateral side when the contra-lateral hemisphere was irritated. No convulsions were produced on irritation of the motor area when the optic thalamus, hypothalamic region and pyramidal paths of one side were destroyed.

Bilateral injury of the tegmentum and pyramid at the level of pons appeared to wholly interrupt the results of cortical irritation. Unilateral division of the tegmentum, with partial or complete injury of the homo-lateral, or of both pyramids, at any level of the crus cerebri between the thalamus and proximal end of the vagus nucleus, arrested the possibility of generating upon the operated side epileptic convulsions in the contra-lateral extremities, while direct irritation of the tegmentum led to persistent tonic contractions in contra-lateral extremities. With intact cortex and partial preservation of conducting paths to the cord isolated spasms may be produced in contra-lateral extremities by faradizing either hemisphere; but after injury (unilateral) of the tegmentum these contractions cannot be evoked. Hence we infer, with Bischoff, that the tegmentum must be a collaborator in the irritation which produces epileptic paroxysms, although from his experiments one is forced to admit that the part played by the tegmentum is relatively small.

Prus has reviewed the literature of experimental research into the functions of the corpora quadrigemina and publishes his investigations upon dogs in this connection. After careful and painstaking investigation he has educed some very valuable evidence in regard to the origin of tonic and clonic spasms, the essential features of an epileptic convulsion. Stimulation of the anterior corpora quadrigemina induced tonic spasm and true epileptic attacks. Prus applied cocaine to parts under investigation to avoid reflex action, which might tend to confuse the experiments. Stimulation of the posterior quadrigemina was followed by clonic spasm. He therefore infers, as do Bischoff and Hering, that the corpora quadrigemina contain motor centers which, under irritation in the anterior part, produce tonic cramps and in the posterior part clonic spasms. Prus experiments also elicited many other



interesting facts not especially bearing upon the subject of epilepsy.

Hering, in an equally interesting series of experiments upon dogs and apes, concludes that all tracts through which muscular contractions may be brought about may also serve for muscular relaxation. Specific inhibitory paths (like the vagus inhibitory) or inhibitory centers have not as yet been proven to exist.

It should also be said that with reference to the cerebral nervous system and the musculature of the body no specific inhibitory paths have as yet been demonstrated, and the probability that such exist is small, for there would then be required a double number of nerve fibers to preside over contraction and relaxation of muscles. Inhibition can be explained without invoking the agency of special fibers for that purpose by the theory that it depends on some underlying alteration in the condition of nervous or muscular tissue; consequently, inhibition is to be explained by physiological and pathological research and not by anatomy. Neither are there demonstrable specific paths for the production of clonic spasms (cortical epilepsy), as all paths subserving movements in general can be employed in the production of clonic spasm. But as all paths do not possess the same degree of irritability, Hering logically infers that clonic spasms may be more easily produced through the pyramids than through other paths.

A very important fact has been determined by Hering's experiments, namely, that dogs and apes appear to show a difference with regard to "corticofugal" paths. In apes the pyramids appear to play a greater rôle in the induction of isolated movements of the contra-lateral extremities (whence it is to be inferred that in mankind a still greater part is played by the pyramids). On the other hand, the dog has an isolated contra-lateral path through which movements can be induced; and in the ape the contra-lateral path can functionate only in connection with the homo-lateral path. Other minor differences between the ape and dog were in evidence. The author is still prosecuting his studies with special reference to the bearing of these results upon human epilepsy. Further report will be awaited with great interest.

## THE EPILEPTIC PREDISPOSITION.

Studies upon the epileptic predisposition have been made by Joffroy, in studying the relationship of alcoholism and absinthism to epilepsy. He calls attention to the marked difference between the infectious diseases like measles and those requiring a special soil for their propagation, such as tuberculosis, gout, obesity and the so-called neuroses. The underlying factor of the latter class is degeneracy.

Of the various kinds of poisoning alcohol and absinthe are most advantageous for this study. Joffroy agrees with Féré that alcoholic and absinthic epilepsy are essentially like true epilepsy at bottom—that is, due to heredity. The statistics of Martin show that 304 children (infants) of alcoholic stock 48 died of convulsions, 144 died of indifferent affections and 112 survived. Of this number the huge total of 60 cases became epileptic. Thus 108 out of the original 304 became victims of convulsive affections (*Gaz. des hôp.*, April, 1879).

Whether it is alcohol, puerperality, digestive disorders or infectious diseases, etc., it is always a poison acting upon a hereditary or acquired predisposition. Degeneracy is held to underlie neurology and psychiatry and implies inferiority, a lessening of the power of resistance. This inferiority is such that it can never be made up, and therefore leads Joffroy to say, with Esquirol, "once a degenerate always a degenerate," and epilepsy is *per se* a disease of degeneracy and therefore incurable. As a passing comment we may say that many cases do recover notwithstanding, which leads us to still study and more carefully scrutinize the peculiarities of the epileptic predisposition.

Hochhaus has put forward the premature calcification of the vessels as a cause of epilepsy, and describes a case which occurred in a man of twenty-eight years. The ordinary exciting causes were absent; the man died of status epilepticus; autopsy proved the lesion in the left central convolution and cornu Ammonis. The resulting malnutrition was the cause of epilepsy and death. The point of most interest is, what caused the early calcification? Virchow has described similar cases due to osseous lesions, but his were never epileptic. Hochhaus' case had no osseous lesion. The lesion described by Hochhaus

can hardly be commonly present in the epileptic brain, otherwise it would not have failed of previous report.

Under title of "Epilepsy of Cardiovascular Origin," Jones and Clinch have presented a study recently. The article first treats of the relationship between heart affections and epileptic attacks. This sketch also gives an account of numerous cases of slow pulse, aortic disease, etc., in association with "epileptic swoon" and other features of epilepsy. Napoleon, for example, had a normal pulse of 40 and suffered from petit mal.

This historic résumé is of much interest and value, but it is too long for extended notice here. The bearings of the subject are numerous. Thus an association of vertigo, syncope and epileptoid attacks is held to be evidence of a degenerated heart (Allbutt). Cardiac syncope certainly shades imperceptibly into petit mal.

A case illustrating the vascular apparatus at fault has been presented by G. Variat, who described a child, nineteen months old, suffering from epileptic attacks caused by congenital cyanosis. The cyanosis was due to a malformation of the heart.

In regard to the increase of intracranial pressure as a cause of epilepsy, Bayerthal has described the action of a long existent meningocele which produced epilepsy, and maintains that if such pressure is long enough continued epilepsy will invariably occur.

A more exhaustive study upon intracranial pressure has been made recently by Nawratski and Arndt, who refer to Kocher's theory, propounded in 1893, concerning the genesis of epileptic attacks; he was able to drain the lateral ventricle and the porencephalic cyst, which communicated with it, and thus cure the epilepsy. From this and other clinical facts he was led to conclude that increased tension of the cerebro-spinal fluid played an important rôle in the genesis of epileptic attacks, the other leading factor being irritability of the cerebral cortex. Kocher and others, therefore, advocated the theory of a drainage of the lateral ventricles.

Nawratski and Arndt's experiments upon three epileptics were conducted only in status epilepticus and serial epilepsy. The rise in pressure during the paroxysms was a *result* rather

than a *cause*, and due to the arrested respiration and disturbance of intracranial pressure. House has recently reattempted to establish Kocher's original thesis.

Epilepsy as a symptom of cortical lesion has been thoroughly studied by Lemos, who stated that cortical lesions seated in the psychomotor zone, or without, especially the latter, may cause not only Jacksonian epilepsy but typical grand mal as well, and that the difference between partial and total epilepsy is purely theoretical.

An exceedingly interesting clinical study upon the family degeneracy that produces myoclonus or paramyoclonus and epilepsy has been most exhaustively presented by Lundborg. His paper first gives a sketch of the entire family stock that produced the fourteen cases of myoclonus and epilepsy; notes upon the seven distinct families from which the cases were recruited; a short résumé of the individual patients; lastly, general consideration of the disease, etc. The stock consisted of several generations of an intensely neuropathic family; three elaborate genealogical tables give the relationship and the neurotic peculiarities of the different individuals.

The following neuroses were especially prominent: tic convulsif, paralysis agitans, simple epilepsy and epilepsy associated with myoclonus. Other more neurotic evidences of degeneracy, such as obesity, tuberculosis, gout, etc., were present. The female sex appeared to predominate markedly over the males, the proportion being even 2 or 3 to 1. The age of nine or ten, or better, the interval of seven to fifteen was by far the most frequent period for the diseases to begin. While heredity played the main rôle (not always by direct inheritance) there were even here many excitants, such as alcoholism, syphilis, etc. There was no evidence of a paramyoclonic epileptic begetting a similarly afflicted child. But few cases were present in which a general neuropathic inheritance was entirely absent. The overwhelming influence of consanguinity in families already predisposed to diseases of degeneracy was most strikingly in evidence in Lundborg's essay. The progeny of the patients who were not childless died of miscellaneous affections (tuberculosis, tubercular meningitis, etc.); a few apparently enjoyed perfect health. In regard to the relationship of the

paramyoclonus and epilepsy, some cases of epilepsy developed before myoclonus, while in other instances the myoclonus preceded the epileptic convulsions by one or two or three years, and possibly, in one or two cases, as high as eleven and twenty years. Lundborg believed that the development of myoclonus upon epilepsy stamped the individual as hopelessly degenerate and therefore incurable.

Taking this entire stock together, we find the following:

|                  |                                  |   |
|------------------|----------------------------------|---|
| 1st gen. (Per P) | healthy?                         |   |
| 2d gen.          | healthy?                         |   |
| 3d gen.          | healthy?                         |   |
| 4th              | Paramyoclonus and epilepsy ..... | 6 |
|                  | Tic convulsif .....              | 2 |
|                  | Paralysis agitans .....          | 2 |
| 5th              | Paramyoclonus and epilepsy ..... | 4 |
|                  | Plain epilepsy .....             | 1 |
|                  | Tic convulsif .....              | 9 |
|                  | Paralysis agitans .....          | 3 |
| 6th              | Paramyoclonus and epilepsy ..... | 4 |
|                  | Plain epilepsy .....             | 5 |
|                  | Tic convulsif .....              | 0 |
|                  | Paralysis agitans .....          | 0 |

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36

Numerous other neurotic peculiarities are incidentally mentioned: idiocy, imbecility, paralytic dementia, suicide, etc.

While the epilepsy and paramyoclonus were undoubtedly closely associated, it could not be determined whether either one stood in any direct causal connection with the other, which is similar to the conclusion arrived at by Garnier and Santenoise in the case reported by them, yet it is practically established in Lundborg's study that epilepsy is particularly liable of occurrence in the great majority of degenerate families.

In connection with paramyoclonic epilepsy, the observations of Bechterew are of interest. He describes chorea-like convulsions in an epileptic. The association was not a coincidence. The choreic twitchings became more intense as the time for a paroxysm drew near, and after the epileptic attack had passed by the choreic movements ceased for a considerable period. The epileptic attack itself seemed to be an exacerbation of the ordinary choreic movements plus loss of consciousness; the chorea, of course, could not be held to stand in any immediate causation to the epilepsy.

## TOXICS AND AUTO-TOXICS IN THE PRODUCTION OF EPILEPSY

Under this head we find most of the present day studies of the immediate excitants to epileptic convulsions. As to the definite poisons, we find a thesis by Bucelli, who states that alcoholics often become epileptics and *vice versa*. He also puts forward an alcoholic equivalent for epilepsy in those patients having a sudden impulsion of dipsomania. This is not to be confounded with true dipsomania, as the act is more automatic and the desire perfectly irresistible. Of 226 alcoholic epileptics studied by Bucelli, only five were due solely to alcohol, that is, the attacks in five appeared after indulgence in alcohol in perfectly sound persons. Ballet believes that alcohol is less frequently a potent agent in the causation of epilepsy than absinth and cocaine. He nevertheless quotes many observations that epileptics leaving asylums have attacks almost immediately, due to indulgence in alcohol.

Bourneville and Dardel and Dide have recently reported many cases in which typhoid has excited the occurrence of epileptic attacks; it is believed that the fever stops the brain development and the retrograde process gives origin to epilepsy. The rôle of typhoid in this connection must always be decidedly secondary to scarlet fever as a toxic agent causing epilepsy. An unsigned article in the *Allg. Wiener med. Zeitung*, after carefully surveying the whole field of present day research in epilepsy, puts forward gas poisoning as a cause of genuine epilepsy. An illustrative case is reported in full; all the pathological findings in similar cases on record from the same cause were reviewed, and a softening of the corpus striatum and inner capsule was found in all. Experiments showed that vascular alterations similar to lesions found in these autopsies could be produced; it was, therefore, believed by this author that permanent damage of the finer vessels of the brain might result by gas poisoning. This "chemical epilepsy" may not be dissimilar to the toxic epilepsy by alcohol which has already been exhaustively and carefully studied.

In the study of the morbid anatomical peculiarities and the toxics generated by them as a cause of epilepsy, the work of Ohlmacher is in evidence. He has published a number of articles upon the subject. Careful microscopical and macro-

scopical study have been and are still being made by him upon all epileptics dying in the Ohio Hospital for Epileptics. He has found eight out of eighteen cases in which there was a persistent thymus; a surprisingly large number, and altogether too large to be explained by coincidence. In the eight cases subjected to autopsy there were found present, in addition to enlarged and persistent thymus, a pronounced enlargement of the intestinal and splenic lymph follicles; more or less hypertrophy of the lymphatic glands and lymphadenoid follicles of the tongue, larynx, trachea, esophagus, and even stomach. The arteries were narrowed, fat was abundantly present and there were osseous changes indicative of old rickets. Every case did not exhibit these peculiarities, the thymus lesion being the only constant one present. These patients were below middle life, and at least six of the eight had idiopathic grand mal. Four had periodic mania; three cases were found dead in bed, having been previously in good health. The subjects of thymic asthma and thymic sudden death in children are well known. The enlarged and persistent thymus is also a part of the "status lymphaticus," or lymphatic constitution. Ohlmacher holds that the same lesions were found in the two states, at least in the eight cases upon which autopsies were made; in addition, peculiarities in the arteries and bones were also found. It remains to be seen whether or not this status lymphaticus is one of the factors at the bottom of epilepsy.

Ferrarina refers to the various examples of neuroses of hepatic origin, and adds a case of apparent hepatic epilepsy. The boy was first epileptic at the age of eight years; the type was classic with later equivalents of ambulatory automatism. The patient was first seen at the age of seventeen. He was somewhat jaundiced; between fits he had urobilinuria, and after a fit biliary pigments appeared. The entire digestive tract was in bad condition. He was placed upon purgatives and milk regimen. The seizures and hepato-intestinal symptoms improved at the same time; urobilin and bile pigment left the urine. Axenfeld (1895) has shown that bile may excite the cerebral irritability when injected into the carotid; and Bickel (1897) has corroborated this. Unfortunately no mention is made of the hereditary factors in this case, a point always es-

sential to determine the relative severity of the toxic agency and its true rôle in the production of the epilepsy.

In the multiple studies upon the autotoxic element in epilepsy, Fitcher, of Johns Hopkins, attacks Rachford's paraxanthin theory in the *St. Paul's Med. Journal*, Sept., 1899. He first gives the chemical nature and relationship of the leucomains, of which group xanthin and paraxanthin are members, and states that paraxanthin acts as a tetanizer to some of the lower animals. Rachford's original paper on leucomain poisoning appeared in 1895; he attributes to the agency of paraxanthin a migraine, an epilepsy, and a gastric neurosis. Rachford quotes largely from Salomon, whose results he is able to confirm. He also thinks that Haig's "uric acid epilepsy" is of the same nature as his own paraxanthin epilepsy; but he holds that uric acid and the urates are not poisonous.

Rachford's views have recently been re-affirmed by their author, hence his older views need not be repeated in this connection. Fitcher next proceeds to adduce evidence against the correctness of this leucomain theory. In 1889 Denis and Choupe found that the toxicity of the urine was normal in epileptics. In 1890 Féré found that the preparoxysmal urine was hypertoxic, and much more toxic than the post-paroxysmal urine. Voisin and Peron found that the paroxysmal urine was hypotoxic; during the paroxysm the toxicity increased while the postparoxysmal urine was hypertoxic. Marret and Bose in 1896 found that the urine of epileptics is in general hypotoxic, and this lowered toxicity is most marked after the paroxysms and between seizures. The urine of the pre-paroxysmal period is relatively hypertoxic, and the normal toxic coefficient is lowered immediately after the fit, the toxicity being reached some hours after the paroxysm has occurred.

In attempting to harmonize these discrepant views, Fitcher states that the consensus of opinion appears to be that the urine of epileptics is hypertoxic about the time of the attacks, its toxicity being much greater than during the inter-paroxysmal period. Thus far these studies of urinary toxicity appear to favor the autotoxic theory of epilepsy.<sup>1</sup>

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<sup>1</sup> The toxicity of the urine will be mentioned again under examinations of secretions in epileptics.



During the present year Pfaff and Putnam attempted to disprove Rachford's claim that paraxanthin is the cause of migraine, incidentally touching upon epilepsy in this connection. They claim that paraxanthin is found in an equal degree in the healthy and unhealthy, the inference being that Rachford's mode of analysis is fallacious.

Deutsch, in an article on auto-intoxications, describes a peculiar case in which epileptiform convulsions were present in the midst of acetonemic coma. Deutsch's patient presented the picture of genuine status epilepticus. As far as could be determined, the patient was free from any congenital or acquired taint. The author chooses to call the affection acute acetone poisoning, and borrows the expression "acetonic epilepsy" from v. Jaksch (1888). He thinks it possible that studies of the source and nature of acetonemia might throw some light on the subject of genuine epilepsy. In this connection Deutsch mentions a case of epilepsy which appeared to be due originally to sudden fright (fall in the water); yet an autopsy two years later revealed a cyst in the brain. He quotes Kranisky's theory advanced in 1897, that epilepsy was due to the periodical formation of carbonate of ammonia.

Beside Deutsch, von Jaksch, Wayner and others have described cases of acetonic epilepsy.

Weber in certain cases believes also that ammonia carbonate produced from urea causes epilepsy. Bond in many cases in which paraxanthin or xanthin urine would be excluded found leucomain poisoning. Errors of diet appeared to play the rôle of genesis of attacks. Bond states that it is precisely the idiopathic cases of epilepsy in which gastro-intestinal irritation occurs most frequently, and it is equally certain that proper treatment directed towards overcoming these factors shows us the best results.

In regard to the vasomotor origin of epilepsy which Gowers has stated is "alike unnecessary and unproven," not much has been done recently. The present status of the matter has been very well set forth by Binswanger in his monograph upon epilepsy, appearing in 1899, which gives a historical sketch of the attempt to identify vasomotor phenomena with epileptic at-

tacks. The literature of the subject extends back to the time of Astley Cooper.

After a careful reading of recent literature upon the subject, we would conclude that the problem of the vasomotor relationship and arterial pressure upon the production of epilepsy is still to be solved. It appears in all the numerous observations and experiments that the cortical discharge takes effect upon the peripheral vascular territories rather than upon cortical or vasomotor medullary centers. In fairness to the believers of the vasomotor theory it may be said that there is a small number of cases of epilepsy which may be ascribed to vasomotor disturbance, especially in those cases where the entire vascular system is involved. This is not only true of grand mal but also of petit mal and psychic epilepsy. In those cases there is turgescence of the skin, profuse sweating and dicrotic pulse. In such cases a sharp rise of temperature is usually found.

We conclude with Binswanger and Bechterew that the only type of epileptic convulsion in which the medullary vascular center is primarily involved is in the reflex species which are certainly exceedingly rare.

#### MANIFESTATIONS OF EPILEPSY.

In reviewing the different manifestations of epileptic phenomena, we find the psychical equivalents have received a great deal of attention; of late, several voluminous works have appeared. Bombarda has published a small volume containing a study on the sleep states in epileptics, consisting of nightmare, starting in sleep, pavor nocturnus or night-terrors, seminal emissions and erotic dreams without ejaculation of semen. These he holds are nothing more than equivalents of petit mal epilepsy. He therefore believes a nocturnal pollution to be a true attack which is quite in accord with the views of Zuccarelli and Hamilton on the subject, but which is generally, and it appears to us justly discredited by conservative neurologists.

Many cases of retarded epilepsy have been reported of late, principally studied by Guillon and Allen, but the studies upon them have not especially enriched our present day knowledge of the essential nature of epilepsy. Accompanying the vascular

changes generally found there is almost a marked predisposition.

Toulouse and Marchand and Péon have recently reported cases of general paralysis in youths who were also epileptic; but as juvenile paresis is almost always luetic (at least it is so in Mott's opinion, by whom the subject has been recently thoroughly and exhaustively studied), it seems doubtful if the cases were other than juvenile general paretics in which epileptiform convulsions were very prominent.

Féré has held for some time that laughter may be an equivalent in many epileptics and has recently reported one unique case of this kind of epilepsy. Equivalents as well as autoxics in epileptics appear to be the *bête noire* of many present day investigators in epilepsy.

Lemoine has reported the gastric forms of epileptic paroxysms; in most instances such cases appear to be the aborted gastric or epigastric aura of true epilepsy, and not deserving of special classification.

Bandropadhia reports a possible case of tetanoid epilepsy first described by Prichard. The case was apparently cured by application of iron rings on the toes where the fit began with a distinct aura. The fact of the cure in the case alone makes one look askance at the diagnosis of true epilepsy.

Von Bechterew, in a careful study of epileptic and epileptoid paroxysms of morbid fear, concludes that such are not infrequent in epilepsy; they may exist as an aura or alternate as equivalents with comitial paroxysms. In rare instances minor epilepsy may exist as paroxysmal fear alone. As a rule, loss of consciousness or vertigo do not occur. This form of epilepsy, as has been determined by many writers, is refractory to the usual anti-epileptic treatment. Bechterew insists on the essential difference between this form of epileptic fear and that accompanying neurasthenia. Féré also deals quite exhaustively with the subject, and details cases somewhat similar to those of Bechterew.

Féré describes and discusses at considerable length three cases of narcolepsy or paroxysmal sleep as equivalents of idiopathic epilepsy. The cases are rare, unique and quite without rational explanation as yet.

Higier describes a case of periodic paralysis not consecutive to convulsions. He refers to the experiments of Sherrington (1893) and Mislawski (1898), to the effect that if a given portion of the cerebral cortex is electrically excited, some muscles act spasmodically while others are relaxed; if the flexors contract, the opposing extensors are relaxed. He therefore concludes that if the intensity of the cortical irritant is of low degree paralysis may result instead of convulsion. This may in fact be demonstrated in part by experiment.

Cestan and Le Sourd discuss at great length the clinical value of the "toe phenomenon" of Babinski, and give the result, among other diseases of the nervous system, of examination of a number of cases of epilepsy (the toe phenomenon is an extension of the toes instead of flexion upon pricking the sole of the foot). Babinski maintained that there was a close clinical connection between the toe phenomenon and "spinal epilepsy," mainly because the toe phenomenon showed as the first symptoms of pyramidal involvement in disease of the nervous system. Cestan and Le Sourd, therefore, hold with Babinski that in partial epilepsies dependent upon organic changes in the pyramids the toe phenomenon is of great importance in diagnosis. This is especially valuable when we remember that even now many organic lesions upon which the so-called idiopathic epilepsies really depend go unrecognized because of the great difficulties which surround their detection.

Hughlings-Jackson has reported and described "uncinate epilepsy" cases in which there is slight asphyxia, subjective odor, chewing movements and flow of saliva, and an association of the so-called "dreamy state" in this type. Autopsy on one typical case proved the lesion true to the diagnosis before death (Jackson and Coleman).

J. W. McConnell also describes several cases of transient paralysis as epileptic equivalents, the cause of these epilepsies he attributes to auto-intoxication.

Several articles have recently appeared upon the type of epilepsy accompanied by, or consisting of, apparently purposeful movements of locomotion, such as running forward, backward, sidewise; spinning and rolling before convulsions or while in the fit, or even without a fit. Wittner in an article

upon chronic diffuse post-epileptic encephalitis relates a case of procursive epilepsy in which, contrary to the title, he proves it to be secondary to organic mischief as above noted in the title. Bourneville has long believed that procursive epilepsy gradually produces encephalitis.

Ziehen in a thesis which contains his well known hypothesis based on experiments that clonic spasms are cortical, and tonic spasms of infracortical source, holds that procursive movements are infracortical in origin. In a more recent article of last year, Schuster and Mendel reported four cases of procursive epilepsy. Contrary to usual experiments, bromides were of signal use in their treatment. These authors, without the privilege of autopsy upon any of their cases, but with apparent logic, believe that this phenomenon of epilepsy is but one phase of true epilepsy.

A bizarre and rare form of epilepsy, long known, although but little commented upon of late, is the so-called attacks of hunger or states of hunger in varied associations with epileptic phenomenon. Hunger-states seem closely allied to attacks of fear described by Bechterew and others. The hunger-state should not be confounded with boulimia, a singular craving for food, common in epileptics and other neurotic people, nor with pica, a craving for unnatural food, seen in some pregnant women. Féré has reported several cases of the "hungry evil" in which it existed both as aura and as an equivalent. In this latter phase it alternated with classic attacks of epilepsy.

Two studies have been made recently in regard to the analogy of epilepsy and eclampsia; one by Dide in pointing out their similarities of heredity and auto-toxic bases, cites a case in which eclampsia gave origin to epilepsy; cases illustrating the above point have long since been placed on record. The infrequency of such an association leads one to look for other factors favoring the relationship. Under the exceptional circumstances of eclampsia causing epilepsy, Chambrelent, in reviewing the whole subject of pregnancy and epilepsy, found that some fifty epileptic women had been carefully observed while pregnant. In over half of the number the epileptic condition was benefited. Having decided on the whole, pregnancy exerts a favorable influence upon the epilepsy, Chambrelent

finds that the epilepsy has no effect upon pregnancy. Even a status case did not abort. A great number of children born of epileptics have convulsions and some die from them. Probably in time statistics will be forthcoming in which some relationship may be found to exist between cases which became worse from pregnancy, and those who bear children who in turn develop convulsions. The subject is not only one of considerable scientific interest, but also of much practical importance.

It has long been a study to ascertain why epileptics have their attacks at night only. In such cases many theories have been advanced to explain the phenomenon which have been detailed at length by Pick, but none has met with general favor. It is generally conceded from all statistics that the hours 9 P. M. and 5 A. M. are the ones in which patients most frequently have attacks. Neither circulatory disturbances (Féré), nor depth of sleep (Kohlschutter, Czerney, Howell) seem to be sufficient to cover the explanation necessary. In fact, in regard to the latter theory, the frequency of attacks seems to be in inverse ratio to the depth of sleep.

Some work far from harmonious has been done upon the toxicities of the blood, urine and sweat, in recent years. Lui claims to have demonstrated marked variations in the alkalinity of the blood which seemed to have provoked acute psychoses and epileptic paroxysms. Weber maintains after careful observations that ammonia carbonate produced from urea, is the toxic agent which causes many epilepsies. Mairet and Vires infer from test of the blood serum of epileptics that epileptic blood loses some of its normal toxicities and in accord with the results of Herter's experiments they find the urine of epileptics is hypotoxic.

Voisin, in his study of the elimination of methylene blue in epileptics, injected during the attacks and the intervals between, finds a coincidence of his experiments with those of Bar in eclampsia; namely, that the elimination of methylene agrees with the lowered toxicity of the urine after an attack, showing defective elimination of poisonous substances, the evidence of an auto-intoxication.

In studying the relationship of migraine to that of epilepsy, Rachford thinks that paraxanthin ought to be found after

paroxysms of epilepsy; but many observers of late have found it is infrequently the case, paraxanthin epilepsy being much more infrequent than paraxanthin migraine.

Mavrojannis concludes after a most painstaking series of observations that the sweat of epileptics has but feeble toxic power, much less than Cabitto found. Injections of sweat in rabbits produced extension of the vertebral column only, not convulsions; but even extension of the vertebral column was not obtained with the sweat of normal subjects.

Uniformity in the presence or absence of post-epilepticalbuminuria is wanting; authorities are about equally divided as to its presence and absence after paroxysms. It is generally held with Pio Galante that the albumin of the post-paroxysmal state, when found, is due more to the respiratory arrest than to the increased vascular tension in the fit. Many theories and many supporters of each theory still hold prominent place. The great majority of neurologists still deny its habitual presence in the post-paroxysmal state of all epileptics.

As to post-paroxysmal exhaustion in epilepsy, it is manifested in many ways and of late has been well studied. Exhaustion-paralysis is shown in the skeletal muscles, ocular exhaustion, aural exhaustion, exhaustion of cutaneous sensibility and the rare phenomenon post-epileptic dysesthesia, recently studied by Féré. Post-epileptic pain and sensitiveness is a rarer phenomenon than anesthesia, which in turn, is of quite uncommon occurrence. The motor phenomenon of post-paroxysmal exhaustion is the most frequent of exhaustion phenomena. All the exhausted states may occur isolated or in varied combinations. They are subjects which deserve very close study and attention, as they are not infrequently indices to the brain lesion causing epilepsy.

Féré with others, has recently urged all the different forms of post-epileptic exhaustion phenomena as equivalents as well as sequella of paroxysms. There is still much to be done to solve the problem of relationships of equivalents and epileptic crises.

Rennie has recently published an interesting paper on hypotonia in epileptics. For many years hypotonicity as a post-epileptic sequel indicating the exhaustion has been written

upon by continental physicians under the head of post-epileptic rigidity, and contractures as paralytic equivalents. Rennie reports three cases highly illustrative of the title of his paper.

*(To be continued)*

- 58 RECHERCHES THERMO-ÉLECTRIQUES SUR LE CERVEAU D'UN ÉPILEPTIQUE. Dr. G. Mirto (*Arch. italiennes de biologie*, Tome XXXII, Fasc. II, 26 Février, 1900; pp. 335-340).

G. Lombardini, an epileptic, aged fifteen years, has a gap in his skull (caused by a fall in early infancy), irregular in shape, nine centimeters by four, and situated, with its long axis antero-posterior, in the parietal region. Above and behind it is over a small part of the parietal ascending convolution; behind it involves almost the whole of the superior parietal convolution and the highest portion of the inferior convolution. Convulsive attacks occur at intervals of days or of weeks, while attacks of epileptic vertigo are more common. He is mentally feeble and his vision is very defective.

Dr. Mirto studied the thermal changes in the cortex under various conditions. He employed a thermo-electric pile of forty couples of bismuth and antimony plates connected with a mirror-galvanometer of such sensitivity that a rise of one degree in temperature caused a movement of about 200 mm. on the scale. This was about the largest deviation he had ever obtained from cerebral changes, the instrument being applied to the hair-covered scalp.

The observation here recorded was made while the author was watching the scale, and consisted of a deflection of 42 mm. in the positive direction, corresponding to a rise of temperature of  $0.2^{\circ}$  on the surface of the scalp. It reached its maximum in 50 seconds, and in 115 seconds decreased to 8 mm. During this time the subject had undergone an epileptic seizure without convulsions; he was pale, with closed eyes, and in a state of semi-stupor, and had passed his urine.

Thus is confirmed by accident under ideal conditions the conclusions of Mosso arrived at from experiments on dogs, that an epileptic seizure is accompanied by a cortical rise of temperature independently of muscular innervation, for in this case no convulsions occurred. Dr. Mirto concludes as a corollary that the heat-producing process of the cortex is or may be independent, both of motor discharge and of psychic concomitance, as Mosso had already maintained.

G. V. N. DEARBORN.

- 59 SUR L'IRRITABILITÉ. Note by Prof. A. Stefani (*Arch. italiennes de biologie*, XXXII, III, 26 Février, 1900, pp. 439-450).

In this "note" Professor Stefani reviews the theory of irritability elaborated by Hering and published in 1889, basing the phenomena of irritability on automatic changes in the nutrition of the tissue, and practically claims that as early as 1879 he himself had published a similar doctrine on this subject.

G. V. N. DEARBORN.



PHILADELPHIA NEUROLOGICAL SOCIETY.

February 26, 1900.

The President, Dr. William G. Spiller, in the chair.

TWO CASES OF TABES DORSALIS IN NEGROES—HUSBAND AND WIFE.

Dr. A. P. Francine presented a husband and wife, of negro blood, with tabes. The man was 48 years of age. He positively denied the presence of white blood in his family. He had had a chancre when 17 years old, and was married to his present wife in 1888. Five years ago vision began to fail. The diagnosis of tabes was made on account of ataxia, shooting pains, numbness in the soles of the feet, girdle sensation, lost knee-jerks, Romberg's sign, Argyll-Robertson pupil, optic atrophy, etc.

The wife was 52 years old. She denied the existence of white blood in her family. She had had two children by a former husband, but had not been pregnant in her present marriage. About a year ago she began to suffer from sharp pains in the legs and trunk, and difficulty in walking. The diagnosis of tabes was based on these symptoms and on dimness of vision, Argyll-Robertson pupil, girdle sensation, partial double ptosis, lost knee-jerks, Romberg's sign, etc.

Dr. H. J. Berkley, of Baltimore, said that he had not seen many cases of tabes in the negro and considered the disease an extremely rare one. On the contrary, the allied condition of dementia paralytica in the negro is occurring in ever greater frequency, until at the present time, in Baltimore, the proportion of cases between negro and white paralytics is about the same in proportion to the general population. Before the war, dementia paralytica was unknown in the black race. It has only been since they have been subjected to what Krafft Ebing calls "syphilization and civilization" that the disease has appeared among them.

Dr. Charles W. Burr said that the man had been referred to in several papers on tabes in negroes read before this society. It is interesting that his wife now has the same disease.

Dr. J. Madison Taylor had known this man for 5 or 6 years. He considered it an error to regard these cases as pure negroes, as any one familiar with the negro would at once detect characteristic evidences of admixture in both cases.

Dr. F. Savary Pearce said that five years ago he had reported to this society the case of a white man who had had a sudden apoplectic attack proving later to precede locomotor ataxia. This man's wife also had locomotor ataxia which had existed for thirty years previous to the husband's disease. Under these circumstances it seemed that syphilis could be ruled out of the etiology. While it is possible that a toxin might be transmitted from the man to the woman through insemination, one could hardly see how a toxin could be transmitted from the woman to the man in a case where there are no secondary lesions.

In a study of 194 cases which he had made in May, 1898, there were only two cases observed in the pure-black race, a proportion of

about one per cent.; showing that the disease must still be regarded as rare among Africans. It is interesting to note that the colored man reported by Dr. Francine was one of the cases that for a time was much stimulated by testicular fluid injections (See Transactions Medical Society of Pennsylvania, 1896).

Dr. William G. Spiller said that the statement of Dr. Berkley that dementia paralytica is increasing among the colored people is of interest. It is the belief of many that dementia paralytica is almost invariably a syphilitic disease. In a recent discussion on this subject by the Pathological Society of London many of the speakers held this view. If both tabes and paralytic dementia are syphilitic diseases, the increase of dementia paralytica, and not of tabes, among the negroes is very peculiar.

#### TWO CASES OF PARKINSON'S DISEASE.

Dr. Wharton Sinkler showed two patients suffering from Parkinson's disease, in whom there were certain points of interest, and in whom the symptoms were rather atypical. The first patient was a man aged 53; a stable boss by occupation. He had never been unduly exposed to wet or dampness, and had sustained no violent shocks or injuries. About three years ago he began to notice a tremor in the right hand. This increased in constancy and intensity until the present time, and there is now a continuous and excessive tremor of the right hand. There is some tremor of the leg. The tremor involves the fingers, hand and forearm, but the position of the hand is not that which is characteristic of paralysis agitans. The tremor can be arrested by voluntary effort; for example, while grasping the hand of the examiner there is no tremor for several minutes, and the tremor can also be arrested by making some other movement of the hand; for example, patting the leg, as was the case in the patient exhibited by Dr. Spiller at the last meeting. There is no change in the facial expression, and there is no peculiarity of gait.

The other case was noteworthy because of the early age at which the disease began, and the intensity of the symptoms. The patient was a man of forty-five years, an architect by profession. Ten years ago, when thirty-five years of age, he slipped into an opening in the floor of a building under construction, and fell in as far as the hip of one leg. He had to be pulled out, but was apparently not injured at the time. He walked home without difficulty. Several months later he began to observe stiffness in the left leg, and some tremor in the hand. These symptoms continued, and the tremor extended gradually to all of the limbs. About three years ago it was accidentally discovered that there was a prominence in one of the lumbar vertebræ, and on showing this to the physician who was then attending him—a homeopath—an operation upon the spine was urged. A long incision was made, the scar of which

remains, and the surgeon says that he removed a fractured transverse process of one of the lumbar vertebræ. The patient says that, although he was not benefited at all by the operation, since that time he has grown no worse. He presents every feature of a case of paralysis agitans. The expression is mask-like; there is constant tremor of both hands and legs; the flexed position of the hands is characteristic, and there is much rigidity of the body. In attempting to walk there is marked propulsion. The patient can run with great ease, stop suddenly and turn without trouble. He is unable, however, to stand. He can run up stairs two steps at a time with ease. Attempts to write are almost illegible, but if a ball is thrown to him, he catches it with great facility.

Dr. J. Madison Taylor said that he, in conjunction with a highly educated man interested in physical culture, had devised a series of exercises that had relieved some of the most serious features of Parkinson's disease. He did not claim to cure the disease, but to mitigate most satisfactorily the worst disabilities. So far as he knew, this had not yet been done satisfactorily, and he hoped to present these cases to the society for inspection.

#### THE PATHOLOGICAL FINDINGS IN A CASE OF GENERAL CUTANEOUS AND SENSORY ANESTHESIA WITHOUT PSYCHICAL IMPLICATION.

The detailed clinical history of this case, reported by Dr. Henry J. Berkley, can be found in *Brain*, Vol. XIV, Part IV, 1891. A careful search of the medical literature has disclosed no similar instance occurring either before or since the date of its publication.

A synopsis of the clinical record as then published shows: (1) A strong hereditary tendency to nervous instability, the mother, two maternal uncles, two brothers and two sisters having been insane. The father had died of tuberculosis. (2) A syphilitic infection acquired from her husband at the age of twenty-nine years subsequent to which the patient had several abortions, sore throat and falling out of the hair.

For twenty-two years after the subsidence of acute symptoms due to the syphilitic process there was fair health. Then came an attack of acute arthritis after which the patient never fully recovered her former physical condition. Within a few months thereafter her eyesight began to grow dim, there were sudden flashes of light before the eyes, and vision was gradually extinguished, only sufficient remaining to enable her to distinguish light from darkness.

In the early summer of 1889, nearly six years after the rheumatic attack, the patient began to experience a general tingling and formication in the skin of the entire body, which

was shortly followed by several spells of uncontrollable vomiting.

A consensus of the numerous examinations made during the years 1889 and 1890 showed a total loss of thermic, pain, olfactory, gustatory, equilibrium, pressure and weight sensations; almost total loss of the visual sense; and a partial loss of tactile and muscular impressions, muscular sense and auditory perceptions. None of the special senses, or cutaneous sensations remained wholly uninvolved.

Besides these disturbances of the sensory apparatus several other symptoms of almost equal prominence were recorded.

The musculature, while responding to the will, did so in such a feeble manner that the patient was incapacitated from helping herself to any extent. Thus the dynamometer when taken in the hand and squeezed was so feebly compressed that the indicator showed no movement on the dial, though the woman exerted every effort in the trial. Despite this fact, however, both nerves and muscles responded promptly to the galvanic and faradic currents, nor was there anything abnormal noticeable about the quality or time of the reactions.

The cutaneous reflexes were all abolished. The faucial and pharyngeal reflex movements were absent, and a sound could be passed over the epiglottis into the larynx without eliciting a sensation of discomfort or inducing cough. During the attack of vomiting there was no sensation of nausea.

When first examined the knee-jerks were present and normal. The biceps-jerk could also be elicited with some little trouble. The reaction of the abdominal muscles was lost, and the ankle beat was feeble. By the middle of July, 1891, the right knee reflex had disappeared, and the left one was weak. All other reflexes, deep and cutaneous, had been completely abolished. A year later the jerk of the left patellar tendon was found to be extinguished.

Furthermore, with auditory perceptions a progressive dulling could be noted. When the woman was first admitted to the hospital the sense of hearing was fairly acute in both ears. Gradually the difficulty in receiving auditory impressions increased, until finally the voice could not be heard except with strenuous effort.

The ophthalmoscopic examination showed an extensive choroiditis pigmentosa with atrophy of the optic nerves. Only a few vessels could be seen in the retina, and these were of minute size. In both lenses there was a beginning cataract.

Whether the optic nerve atrophy should be considered part of the general disease-process, beginning as it did long before the other symptoms, is somewhat problematical, though in

tabes the same trouble is not infrequently noted as a forerunner of definite symptoms of the disease.

The pupils were at first in a state of mid-dilatation, and responded slowly to light, direct or reflected. They did not dilate on irritation of the cervical sympathetic. At a later stage the pupils became somewhat narrower, but remained sluggish to stimuli.

There were a number of interesting disturbances of the glandular secretions. During the fall of 1889 the mouth was found to be almost absolutely dry, the tongue heavily cracked, the epithelium eroded from its tip and sides, while the entire buccal surface was red and congested. At the same time the secretions of the lachrymal glands had almost ceased, the surfaces of the conjunctivæ being dry and injected. The skin was also dry and devoid of odor. After a course of potassium iodide these phenomena slowly abated, and the secretions returned to a more normal state.

Various unusual sensations troubled the patient at times. Cramps of the gastrocnemii, sensations of burning between the scapulæ, a girdle feeling around the abdomen and neuralgic twinges in the nerves of the extremities were often complained of. Occasionally there were clonic fibrillary spasms of the small muscles of the thumbs.

Attacks of diarrhea, refractory to treatment with drugs, though yielding to a continued milk diet, occurred in the latter part of the winter of 1890. These were on several occasions followed by a protracted vomiting without pain or nausea.

Trophic symptoms in the form of a bulbous appearance of the finger tips and ridging of the nails, together with purpuric spots developed late in 1891.

Throughout the long course of the illness there was never the slightest departure from normal mentality on the part of the patient; no hysterical attacks, no pathological depression or exaltation, and no delusions or hallucinations were ever noted. A slight apathy was the only perceptible change in the mental phenomena, but this was not greater than is frequently noticed in those who have become blind and helpless.

The patient's condition did not materially alter after the record of the case was published. The anesthetic and other symptoms, from the reports of the house physicians to the hospital, remained about stationary, and on the rare occasions on which Dr. Berkley saw her there were no additional phenomena to be noted other than a steady though slow decline of the vital powers. Late in the year 1893, another ophthalmoscopic examination was made by Dr. H. Friedenwald, who

found in the left eye an extensive and typical retinitis pigmentosa, the papilla being blurred and of a dirty yellow color. Few vessels could be seen. In the right eye the clouding of the lens was so profound that the retina could not be seen.

About the middle of May, 1898, Mrs. R. became slightly lethargic, a condition that slowly increased to coma, in which state she died on the 25th of the month.

The autopsy, performed ten hours after death, was distinctly negative, all portions of the central and peripheral nervous systems showing an apparently natural condition. The larger vessels of the thorax, abdominal and cranial cavities, showed scattered atheromatous plaques, but were not considerably thickened. The right middle cerebellar and both posterior communicating arteries of the circle of Willis were congenitally rudimentary. There was some gelatinous thickening of the pia over the central regions of the hemispheres. The optic nerves showed but faint signs of a diseased condition, although the left nerve was a little smaller than the right one. Both kidneys were atrophic, weighing 100 grams each, the loss being principally in the cortex.

After proper hardening for the various Nissl, Weigert, Marchi and other stains, sections of the entire nervous system were made and studied. The results obtained were to a degree remarkable, and for the sake of convenience may be separated into three categories: (1) Lesions appertaining to the blood vessels; (2) those of the proper nerve elements, and (3) those belonging to the membranes surrounding the encephalon and cord.

The fundamental pathological basis for the various nervous phenomena described in the clinical history of the case is as follows: (1) An hyaline-fibrous degeneration of the arterial system existed, which was not confined to the central nervous regions, but was equally evident in the roots of the spinal nerves, and in the skin tissues. The degree of alteration varied from slight thickening of the muscular layer to complete closure of the lumen from hypertrophy of the middle coat of the vessel walls. The morbid change was accordingly not uniform, but reached its maximum of intensity in the vessels of the lower dorsal cord, the meninges of the bulb and cord, and also in certain of the root bundles of the cerebro-spinal nerves. (2) As a consequence of the vascular lesions there were degenerations of an atrophic order in the nerve cells of the gray horns of the cord, more particularly in those of Clarke's column, and in the medulla oblongata. To this alteration in the central nervous substance at least a portion of the various symptoms must be attributed.

Besides the principal lesions there were others of considerable though minor importance.

As an explanation of the numerous symptoms of the case it would appear most reasonable to suppose the existence of a disease process affecting simultaneously both the peripheral and central nervous systems. Assuming that the arterial lesions were of late specific origin—and of this there can be but little reasonable doubt—a chronic progressive involvement of the nutrient channels, here and there leading to marked narrowing, or even to closure of isolated vessels, might certainly have produced just such a train of symptoms as was present. Of primary importance would be the involvement of the arteries of the anterior and posterior nerve roots of the bulbo-spinal system, though the direct lesions of the nutrient supply to the terminal nerve apparatus, which have to be inferred as they could not be definitely determined, would be of equal value. This inference of the implication of the end-apparatus is justifiable since the arterial degeneration in the skin was quite extensive, and whenever there is starvation of a nerve tissue there result pathological reactions which may be manifest in a multitude of ways.

The slowly progressive character of the symptoms is also consistent with the theory of tissue starvation. All the lesions of the nerve cells of the cord and bulb are of this type—atrophy and pigmentary degeneration from malnutrition. Whenever nutrition is at a low ebb, metaplastic granules accumulate in the protoplasm of the cell.

The condition of the optic tracts—in which the lesions are identical with those of other nerve roots except that they are more advanced—would argue that the thickening of the blood vessels was of long standing, and that only when the process had advanced to an extreme degree did any definite symptoms show themselves. This is exemplified more particularly in the state of the cortex cerebri. Though in this region vascular disease was manifest and diffuse, it had not advanced nearly to such a degree as in the gray matter of the dorsal cord or in the adjacent meninges. As a consequence, the functions of the cortex, while not as perfect as in youth, were not reduced to the same low level as those of the cord and bulb.

One pathological fact should be remembered in considering the clinical symptoms, namely, that it is not necessary for a vascular lesion to proceed to such a profound degree as to cause the entire shutting off of the nutrient supply before a nerve tissue will show signs of deviation from its normal functions. With a reduced supply of nutrient plasma, definite manifestations of nervous exhaustion are brought about, and

these are not due to a degeneration of the component portions of the neuron, which is visible in the tissue after death by our present methods of preparation, in the form of morbid alterations of the cytoplasm, axone, or myeline. Long before this stage is reached the entire neuron is incapable of performing its natural functions in an efficient manner, and as a consequence, anesthetics, paresthesias, diminution or exaltation of the reflexes, and dulling of the special senses can be noted. Almost precisely similar results are encountered in advanced stages of progressive paralysis, especially in the syphilitic cases, in which, when vascular lesions of the arteries of the cerebrum and cord have advanced to a profound degree, there is a gradual but progressive dulling of cutaneous sensibilities and special sensations.

Dr. C. W. Burr exhibited a case of general anesthesia.

Dr. F. X. Dercum remarked that while the interpretation which Dr. Berkley had given was exceedingly interesting, it did not seem entirely satisfactory. In cases where there is great vascular degeneration, as in pronounced arterial sclerosis, the sensory symptoms found in Dr. Berkley's case do not occur, and therefore the symptoms could hardly be ascribed to the paucity of the blood supply.

It seemed to him that this case would suggest the presence of a toxin. The eccentric position of the nucleus and nucleoli, among other things, would suggest this, and the changes in the blood vessels and kidneys would be in keeping with such a view.

Dr. Joseph Sailer said in reference to the statement that double nucleoli in a nerve cell were indicative of an irritative lesion, that he had repeatedly found these double nucleoli, and in looking up the subject, found that they had been noted by others in cells presumably normal.

Dr. W. G. Spiller said that a few cases of general anesthesia, and some with necropsy, had been reported. He had observed a case of this character in the service of Dr. Dejerine at the Salpêtrière, and had been permitted by Dr. Dejerine to study the specimens from another case reported later by Dr. André Thomas. In the latter case no such arterial disease as in Dr. Berkley's case was observed. There was, however, an extraordinary condition of the central nervous system: the cerebellum and spinal cord were small, and the posterior columns markedly degenerated. Dr. Thomas ascribed the general anesthesia to hysteria.

Dr. Spiller thought that if the general anesthesia in Dr. Berkley's case were produced by the vascular lesions, we should expect to see general anesthesia in other cases of vascular disorder, as in syphilis. In some such cases there is extreme degeneration of the blood vessels with great impairment in the nutrition of the tissues, yet we do not find persistent and general anesthesia in such cases. Dr. Berkley's report was a most important and interesting one.

Dr. Henry J. Berkley said that while a number of other cases of general cutaneous and sensory anesthesia had been reported, there were none in which the pathological examinations made disclosed the same anatomical findings as in his case. His case stood in this respect isolated in medical literature.

With reference to double nucleoli, he said that these were found normally in some of the lower animals. He doubted the correctness



of the statement that they are found normally in the human being. In many thousand sections he had never found them except where there were evidences of an irritative process.

Dr. Berkley could not agree with the suggestion of Dr. Dercum as to the condition being due to a toxic cause. While the atrophy of the kidney and of other tissues might induce the presence of more or less toxic material, yet in a case where there is such immense alteration of the blood vessels, and these occurring much better developed in one locality than in another, he did not think it necessary to seek any other cause for an explanation of the symptoms. In any tissue that is half starved, one always gets nervous symptoms. Lesions of the media are more liable to produce a symptom-complex corresponding to the phenomena in the present case than when there is alteration in the other coats. When the hypertrophy of the middle layer is very great it almost completely stops the circulation of the nutrient plasma.

#### BRAIN FROM A CASE OF CONGENITAL ABSENCE OF THE VISUAL TRACTS.

Dr. W. G. Spiller exhibited the brain from an idiot, supposed to be about twenty-two years old. The palpebral fissures of the patient were very small and no indications of an eyeball was seen in either orbit. Not the slightest opening in the cranium or the passage of an optic nerve could be found. The optic nerves, chiasm and optic tracts were entirely absent. No external geniculate body could be seen on either side. The pulvinar was considerably smaller than in a normal brain, and no indications of an optic tract passing from it was found. The anterior colliculi of the corpora quadrigemina were of normal size, which would seem to indicate that these structures in man are not an essential part of the visual system. The cuneus on each side was very small, although a calcarine fissure was present.

60. UEBER EINEN FALL VON KATALEPTIFORMER LETHARGIE MIT SIMULATION VON CHYLURIE (Cataleptic Lethargy, with Simulation of Chyluria). M. Rothmann and A. F. Nathanson (Arch. f. Psych. und Nervenkr., XXXII., 1., p. 283).

A very interesting case of hysteria in a girl of 19 is here reported. The patient brought about a condition of seeming chyluria by practicing intravesical injections of milk. Besides this, she was attacked by cataleptic seizures, accompanied with lessening of the area of liver dullness, diminution of urea and presence of ammoniacal urine. Glancing over the clinical history and experimental observations made on the case, the authors came to the conclusion that the attacks were due to disturbances of metabolism in the liver, and especially of the urea-producing functions.

JELLIFFE.

## Periscope.

### CLINICAL NEUROLOGY.

61. "ÜBER SKOLIOSIS ISCHIADICA" (On Scoliosis ischiadica). Krecke (Münchener medicinischer Wochenschrift, 1900, No. 6).

Four forms of this symptom-complex have been described. The homologous, body inclined towards the side of the existing sciatica, the convexity of the curve in the dorso-lumbar portion of the vertebral column toward the unaffected side; heterologous, body to the sound side, convexity to the affected side; and the voluntarily and involuntarily alternating.

Attempts at explanation of the phenomenon have been based upon: (1) Assumption of the easiest position by the patient. (2) Functional disability. (3) Muscular contraction. (4) Muscular paralysis.

The author discusses these different explanations and describes a case of the voluntarily alternating variety, which had come under his notice. The patient was a strongly built man of 33, who, after exposure and wetting, was seized with severe left-sided sciatica. In consequence, upon moving about, he began to incline his body to one side, and for the first six months this was always the left. He then found that by holding fast to some stationary object, he could push his body over to the other side, the lateral curve in the spinal column changing its direction with some suddenness. This movement he had become accustomed to execute quite frequently, changing his position six times during three minutes while under observation. There was a point sensitive to pressure over the left sciatic foramen. When lying on the abdomen the scoliosis entirely disappeared. The patient could bend strongly to either side, but if at the start the body was inclined toward the opposite side, it was necessary for him to go through his regular maneuver of changing sides before he could do so.

It is pointed out that in this case there was evidently no loss of function in the sacrolumbalis, and that a contraction of this muscle could also be excluded.

So to explain it, it is necessary to fall back upon the theory of voluntary assumption of position by the patient.

To relieve the pressure at the sciatic foramen, the patient inclined his body to the sound side, lowering the pelvis on the affected side. Upon so doing, however, the left sacrolumbalis (since Erben has shown that on inclining the body the muscle of the opposite side is the active factor) was contracted, and soon began to exert painful pressure upon the nerves of the back, causing the patient to change sides to secure relief from this last. The pain at the sciatic foramen recurring, he changed back again, and so on. The author thinks that if properly studied, all cases of scoliosis ischiadica would be found to depend upon the assumption, voluntary or involuntary, of a position calculated to relieve certain definite points from pressure.

ALLEN.

62. UN CAS DE TYMPANISME ABDOMINAL D'ORIGINE HYSTÉRIQUE (A Case of Abdominal Tympanites of Hysterical Origin). F. Benoit and R. Bernard (Nouvelle Iconographie de la Salpêtrière, Jan. and Feb., 1900, 13th year, No. 1, p. 57).

A case of a man, normal in appearances, without neuropathic history, either hereditary or personal, who is compelled to give up his work

on account of a progressively developing abdominal tympanites. This tympanites is never spontaneously produced, but is always provoked by one cause—fatigue. Any exercise, as walking, or working at his trade, that of a tailor, produces this condition. When he entered upon his military duties, the fatigue produced by exercise, marching, etc., caused the tympanites to develop to such an extent that he had to be sent to a hospital. The diagnosis in this case lay between hysteria and simulation; the latter was excluded and the former was diagnosed, although all other symptoms of hysteria were absent. This case is a unique one; no similar condition of intermittent and provokable meteorism is to be found in literature. The following résumé of the case and its physiological explanation is given: The condition is one of visceral hysteria, monosymptomatic in type, as it often the case with young subjects. It is caused by a paresis of the smooth muscles of the intestinal tract. The attacks are provoked by effort and cause a paroxysmal meteorism of the intestine. This manifestation is not rare in hysteria, but is most often found in women, particularly at the menstrual period, under the form of a permanent meteorism, causing by its presence embarrassment of the respiration, syncope, and sometimes symptoms of asphyxia. At times, instead of this form, it runs its course under the symptoms of phantom tumors and of localized points of intestinal distension, preceded and followed probably by points of stenosis.

SCHWAB.

63. A STATISTICAL INQUIRY INTO THE PREVALENCE OF EPILEPSY AND ITS RELATION TO OTHER DISEASES. J. W. Russell (Brain, Winter, 1899).

From a series of observations on some 5,000 cases attending clinic for other disease than epilepsy, Russell presents a statistical study on the heredity of epilepsy. He paid particular attention to the finding out of the occurrence of epilepsy in the relatives of those who did not have the disease, endeavoring to ascertain the prevalence of the disease in the community and its relationship to other affections. The proportion of convulsive disorders ascertained was unusually high, 11 per cent. His conclusions are about as follows:

1. Individuals in a state of health approaching normal give a lower incidence of seizures than others.

2. Fifty per cent. and over, of those with definite ailments gave a fairly uniform average of epileptic incidence in the family.

3. A group, including acute bronchitis, phthisis, chronic pneumonia, valvular heart disease, acute and subacute rheumatism, shows a striking increase in the number of patients who have themselves suffered from epileptic convulsions.

4. The most marked epileptic incidence is to be found in the cases of chorea, functional headache, and chronic nervous diseases, in all of which the relationship reaches a high degree.

JELLIFFE.

64. EPILEPSY. Paul (Boston Med. and Surg. Journal, Feb. 1, 1900).

This paper holds that there is no common pathology of epilepsy, and that idiopathic epilepsy is due to an inherent vice of the nervous system. The author believes that 80 to 90 per cent. of all cases are benefited, i. e., have fewer and less severe seizures. The treatment in the outdoor department of the Massachusetts General Hospital is as follows: Each patient has a daily sponge bath, out-of-door exercise, and no excesses or vices are permitted. No meat is given, but eggs, fish and milk are allowed; there is no over-eating, etc., on holidays. All local infirmities, if any, are treated (refraction-errors, phimosis, nasal and aural troubles). Bromide of sodium is the favorite bromide com-

pound, but others are used at times. Arsenic is given for bromide acne. Bromalin, an antiseptic bromide compound, fills an indication stated by Féré (prevention of auto-intoxication). Sympathectomy is at times performed, but no uniform results are obtained from this procedure.

Discussion: Dr. Walton deprecates too close association in the medical and lay mind of epilepsy with infantile convulsions and migraine. As a rule, if an infant's convulsions cease and he lives to be 12 or 13 years old, he is not predisposed to epilepsy. Drs. Walton and Carter have made special researches on this subject. Similar studies of migraine show that a large proportion of epileptics (13 out of 35) have a history of migraine. Nevertheless, there is no evidence that migraine tends to end in epilepsy. The one absolutely constant symptom of grand mal is loss of consciousness, a point of great value in differentiation from hysteria and in legal medicine. Dr. Walton has arrived at this conclusion after much research. A medical man has no right to give a certificate of epilepsy unless he has seen an attack in the person concerned. The fact of having used alcohol on an occasion preceding a fit should tend to weaken the responsibility of epilepsy in the eye of the law; both conditions produce amnesia. Epileptic vertigo is very hard to distinguish from the ordinary variety unless followed by unconsciousness. Epileptics should not be watched, but as far as possible trusted as to going about alone, etc.

Dr. Greene believes in a background of degeneracy, which must be offset by a special mode of life, as in colonies. Epileptics are usually great eaters and need supervision; they are also prone to resort to quack remedies, which they prefer to bromides. The bromides are very depressing in chronic nephritis and arteriosclerosis; they have also been known even in small doses to cause coma, and in other cases hallucinations. One case of Dr. Greene's experience while suffering from bromism was even sent to an insane asylum. If comparatively rapid improvement did not follow bromides, Dr. Greene would discontinue and substitute out-door or colony life. Dr. Greene protests vigorously against cutting meat out of the diet. One case is cited in which a disgusted patient gave up his bromide and went back to meat, yet had not a single attack thereafter for two years. Dr. Greene asks for evidence that meat is harmful in epilepsy.

Dr. Folsom finds many private cases of epilepsy free from evidences of degeneration. He would say that before the period at which dementia sets in there is a great opportunity for amelioration; if dementia has begun not much can be hoped from treatment. He never saw a real cure, but has seen cases go 5, 10, 15 and 20 years without convulsions, and then relapse. In one case the interval was 45 years. He believes in allowing epileptics to regulate their own lives, and gives examples of bad results which followed the contrary course.

Dr. Knapp was unable to trace any connection between irregular or excessive feeding and epilepsy, and therefore does not believe in the auto-toxic theory. He disbelieves in any necessary connection between migraine and epilepsy, but thinks infantile convulsions a possible precursor of epilepsy, and would not trust such a child, for convulsions often lead up to some serious mischief. He has seen cases of minor epilepsy in which consciousness was not lost, and has seen hystero-epilepsy and the true epilepsy in the same patient. He does not believe in cutting out meat and allows it during the day. He is afraid of Flechsig's treatment outside of institutions. He would combine bromides with cardiac tonics as Bechterew recommends. Is skeptical about colonies, because the idea of restriction comes into play and patients often do badly under restraint.

CLARK.

65. CONTRIBUTION A L'ÉTUDE DES RAPPORTS DE L'IMPALUDISME ET DE L'ÉPILEPSIE (Malarial Infection and Epilepsy). Marandon de Montyel (Revue de Médecine, Dec., 1899).

While not in opposition to the recent theory that the infectious diseases exercising a beneficial action upon epilepsy, the latter may be cured or modified by inoculation with the germs of various maladies, the author believes that infection is a two-edged sword, and that its influence is not always beneficial, but sometimes, indeed, injurious. Malaria has by some authorities been held to be a sovereign remedy, so that they had advised the construction of epileptic hospitals in malarial regions, or the sending of epileptics to malarial districts with the view of substituting the latter disease for the former, and then curing it by the administration of quinine. The author reports fourteen cases in which malaria distinctly aggravated the epilepsy, or caused a return of attacks which had long been absent, or even induced attacks for the first time in some neuropathic patients. Therefore, he thinks that causation should be exercised in the treatment of epilepsy by other infections, although it is very possible that in some cases the influence exercised might be a beneficial one.

CLARK.

66. MINOR EPILEPSY. Gowers (British Med. Journal, Jan. 6, 1900).

Tonic spasm is merely a superposition of clonic spasm, and may be considered singly as a subintractant clonic spasm. The slighter forms of idiopathic epilepsy consist of a clonic spasm only, being a slight general rigidity in contradistinction to the clonic spasm of organic epilepsy, and this may be called "medium epilepsy" as standing between the usual classes of grand and petit mal. In minor idiopathic epilepsy loss of consciousness precedes the convulsions; in focal epilepsy the order is reversed and pallor follows minor seizures; then, too, the brain is not anemic in minor epilepsy. In three cases studied with the ophthalmoscope the retinal vessels showed no change. Involuntary micturition may accompany minor attacks and is of great diagnostic value, and if it occur once in the patient it becomes constant; it is much more common in females and may accompany the exclusive tonic attack. True minor epilepsy may occur without loss of consciousness. All possible subjective sensations are met with in minor epilepsy; automatism is always prominent after minor attacks, and from a medico-legal standpoint deserves much attention; hysterical convulsions, too, often follow minor attacks. In differentiating minor epilepsy from cardiac syncope, we must remember that pallor precedes in the latter case; attacks come on slowly and pass off gradually without mental confusion.

Bromides are of much less value in minor epilepsy, and may arrest the major attacks while the minor continue as before. When bromides fail, we may get results from zinc salts, the oxide being the best salt, and is given in from 3 to 10 gr. doses, nausea being readily caused, however. Borax is of inferior utility in minor attacks. The following drugs are of some value in this class of cases: Nitro-glycerine, hydrobromate of hyoscyne, belladonna, Indian hemp and strychnine.

CLARK.

67. CEREBRAL RHEUMATISM. Francis P. Morgan (Phila. Med. Journal, Jan. 13, 1900, p. 116).

Acute articular rheumatism is sometimes complicated by a set of cerebral symptoms so severe and marked as to give their characteristic

stamp to the course of the disease. The term cerebral rheumatism is applied to such cases. In almost any acute disease high temperature may give rise to marked cerebral symptoms—restlessness, sleeplessness, delirium, headache, stupor or convulsions. Some cases of acute articular rheumatism are accompanied by very high temperature and marked cerebral disturbances, such as we should expect from our knowledge of the effect of high fever upon the brain. Therefore, the term cerebral rheumatism usually brings to the mind the thought of hyperpyrexia and its attendant cerebral disturbance.

Morgan cites a number of writers, who seem to think that cerebral rheumatism is due to the effects of a high fever on the brain, and also two who think that there must be some other cause for the cerebral disturbance. He believes (1), that cerebral rheumatism and hyperpyrexia are two distinct and separate complications of acute articular rheumatism; (2), that the symptoms of cerebral rheumatism are not dependent upon high fever, although it may be present; (3), that hyperpyrexia may present symptoms very similar to those of cerebral rheumatism; (4), that true cerebral rheumatism is due to a toxic agent in the blood, which acts on the brain and its membranes, producing the characteristic symptoms. Predisposition plays an important rôle in the production of cerebral rheumatism. The writer cites a case in support of the opinion that true cerebral rheumatism is not primarily due to the effects of hyperpyrexia, and that it may occur without high fever, in which the temperature only once went above 102° F. after the cerebral complications appeared. As to the nature of the toxic agent causing cerebral rheumatism, the investigations and conclusions of numerous writers here cited tend to show that the disease is an infectious process caused by the pressure and growth in the body of the ordinary pus cocci. If this theory be proved correct, all the symptoms of the disease will be easily explained as due to the toxic effects of products elaborated by the pathogenic bacteria. According to Pepper, cerebral rheumatism occurs in about three to four per cent. of all cases of rheumatic fever. In the majority of cases of cerebral rheumatism no pathologic changes are found in the brain or its membranes after death. Occasionally changes are found. When this is the case there is always a strong suspicion that a meningitis complicated the articular rheumatism, and not a true cerebral rheumatism. The symptoms of the disease usually begin with mild nocturnal delirium, headache, disturbance of vision, and subsultus tendinum. Sometimes there is profuse perspiration and vertigo. As the disease progresses the most prominent symptoms are cerebral excitement, great restlessness, deepening at times into convulsions, and coma. Coma is rare, except in the last stage of fatal cases. Usually there is nearly constant wakefulness. The delirium is generally a quiet, talkative kind, with hallucinations, but sometimes is noisy and violent. The hallucinations are usually of space and time and general surroundings. Distrust and ideas of persecution may be present. There may be disturbances of the special senses, with the characteristic symptoms. The motor portion of the brain is affected, and there is restlessness, tremors, choreic jactitation, and even convulsions. The diagnosis is usually easy, as the above symptoms are characteristic. Care must be taken not to mistake the symptoms due to large doses of the salicylates for those of cerebral rheumatism, as they very closely resemble each other. Two cases are reported showing the symptom-picture due to large doses of salicylates. In cerebral rheumatism the prognosis is bad. In 107 cases reported there were 57 deaths and 50 recoveries. The treatment is wholly symptomatic. The condition does not appear to be affected at all by the salicylates.

BONAR.

68. NARCOLEPSY. A CONTRIBUTION TO THE PATHOLOGY OF SLEEP. By D. J. McCarthy (Am. Jour. Med. Sciences, Vol. 119, Feb. 1900, p. 178).

Narcolepsy is a disease which was first described as a peculiar neurosis characterized by an irresistible desire to sleep, occurring suddenly, of short duration, and recurring at frequent intervals. It consists only of repeated attacks of sleep, or of deep somnolence, accompanied by a tendency to fall or sink to the ground. In the literature on the subject, it is found that few agree with this description of the disease or attribute to it the same clinical significance as did Gelineau, who first described it. These recurring attacks of sleep are now considered by different writers to belong to epilepsy; to be due to the derangement of the functions of some of the viscera; or, not to constitute a disease, but to occur only in degenerates, and to be merely a symptom-complex prominently associated with the other phenomena of degeneration. McCarthy holds the latter opinion, i. e., that the attacks of sleep occur in degenerates and are simply the most prominent among other phenomena of degeneration in these cases. He does not consider them a disease *per se*.

He cites four cases illustrating the difference in the clinical picture in the different cases, the difficulty in getting at the causative factors and the importance of the establishment of the underlying disease in the treatment.

The first case was that of a young colored woman, 19 years of age, who for five years had been having sudden attacks of morbid sleep at irregular intervals. The discovery of slight but widespread sensory changes stamped the case as one of hysteria and excluded the possibility of it being one of true narcolepsy.

The second case was that of a white woman, 42 years of age, who had several attacks of prolonged sleep lasting from three days to a week each, and also an attack of hysteria major. Besides the hysteria in this case there was neurasthenia and possibly a beginning melancholia as factors in the production of the sleep. In such cases as this the writer thinks the explanation of the prolonged sleep is that an already exhausted nervous system is attempting to recover from an expenditure of energy which it can ill afford. The vicious, deranged metabolism of such cases should be changed by the elimination of the element of copraemia from the over-loaded bowel. Massage and exercises should be given to alter the tone of the muscles, and to correct the muscle-metabolism, and the entire system should be built up by rest and judicious feeding. There is no reason why the patient should not sleep, but this need not interfere with the feeding of the patient or the massage.

Hysterical conditions should be differentiated as a class from the other conditions of the organism which produce morbid sleep. McCarthy divides the latter into two classes: (1) the epileptoid sleeping states, such as petit mal, epileptic equivalent, etc., and (2) the morbid sleep depending on toxemic states and disturbed cerebral circulation and nutrition. To this class belong the somnolence of uremia, cholemia, etc.; of pulmonary and cardiac diseases; of organic brain disease, and arteriosclerosis; of toxic products from without, such as opium; and the cerebral exhaustion of typhoid and some blood diseases.

He cites a case of petit mal, with attacks of morbid sleep, and also one illustrating the relation between the toxemic states and states of disturbed cerebral nutrition and circulation.

The writer has found nothing in his studies of sleep and its disturbances to support the idea that the condition known as narcolepsy is a distinct neurosis or disease. BONAR.

### THERAPY.

- 69 ZUR INDICATIONEN DER BESCHÄFTIGUNGSTHERAPIE BEI FUNCTIONELLEN NERVENKRANKEANTZEN (On the Indications for Occupation Treatment in Functional Neuroses). Vogt (Wiener-Klin-Rundschau, 1900 XIV., 2 and 3).

After a pretty thorough review of the subject, the author draws the following conclusions: 1. Work as a curative measure is only to be taken into consideration when it is desirable to distract the patient's attention from himself, to furnish a healthy outlet for a desire for activity, and to procure relief from depressive feelings, where this is not being accomplished by the present occupation of the patient.

2. Work is only indicated in so far as it takes into account the existence of any condition of exhaustion and does not tend to produce such a condition.

3. In akinesia algida and in hypochondriasis, occupation therapy has proved of but little use.

On account of the fleeting character of its symptoms, nosophobia needs no work cure.

Occupation therapy is a very important aid in neurasthenias with a tendency to hypochondriacal introspection, and especially when certain physical symptoms remain after a neurasthenia. In imperative conceptions the work cure is of only secondary importance. In severe hysteria occupation may prove a most important curative factor. For many psychopathic individuals work may prove a great blessing.

ALLEN.

70. ERFABRUNGEN IN DER BESCHÄFTIGUNG VON NERVENKRANKEN (Experiences in Employment for Nervous People). A. Grohmann (Psychiatrische Wochenschrift, June 3, No. 8-9).

This article is a short account of Grohmann's experiences in his method of employment therapy in cases of nervous affections. Grohmann's ideas are set forth in his book—"Technisches und Psychologisches in der Beschäftigung von Nervenkranken—Bericht an die Ärzte," which as a new departure in neuro-therapeutics should be read by all neurologists. The conclusions as set forth in this paper are derived from one hundred and six patients and six years' experience. The average duration of the treatment in each case at Grohmann's institution was six months.

The results in general were good enough to convince Grohmann of the correctness of his theory. Some of his failures were due to the unfitness of the patients sent to him for treatment. In almost all patients who remained long enough there was a definite change to be observed, as they became accustomed to the simplified manner of living. The normal degree of fatigue which the patients obtained by means of manual labor made sleep and rest easy for them. In some cases, the habit of masturbation declined considerably; in other cases, a general physical improvement was observed. Alcohol was absolutely interdicted in all cases. Among the most favorable results were some cases of acute insanity in young adults—some cases of imperative ideas in hysterical and imbecile individuals. Especially gratifying was the success attained in several cases where the patients were manifestly



unfitted for the profession which they were following. By a change to a suitable employment a great improvement took place. The article concludes with this sentence: "Altogether I believe that the employment therapy for nervous people is a test of great patience and endurance." Whoever feels that he has the necessary patience and interest will be able to help many people. The method as yet cannot be used in a large way. SCHWAB.

- 71 DES INJECTIONS SOUS-ARACHNOÏDIENNES (Subarachnoidal Injections). A. Sicard (La Presse Médicale, May 17, 1899, p. 229).

The author, believing that Quincke's spinal puncture should be of therapeutic as well as diagnostic value, has undertaken to study experimentally the results of injections into the membranes of the spinal cord and of the brain. In performing these operations he has always exposed the dura mater in order to be certain that the needle punctured it. Various difficulties were experienced in different regions. Intracranial injections were rendered more difficult on account of the resistance and elasticity of the dura mater beneath which the liquid would accumulate in masses. In order to reach between the atlas and occipital bone, it is necessary to destroy a large amount of tissue, and lower in the spinal column there is often danger of severe hemorrhage, and difficulty in keeping the wound clean subsequently. However, as soon as the cannula is properly inserted, the injections can usually be made readily. He found that liquids injected into the subarachnoid cavity were usually absorbed, but somewhat slowly. Various substances were employed, and it was found that in dogs considerable elasticity existed; permitting an injection into a dog weighing 15 kilograms of as much as 200 ccm. of saline solution, without the production of any symptoms. If, however, the injection is continued until as much as 250 or 350 ccm. has been employed, death usually occurs, occasionally with convulsions. Injections of oily substances, such as olive oil, to the amount of 30 ccm. usually produce difficulty and sometimes paralysis of the respiration. Injections of large quantities of air usually produced epileptic convulsions. Small quantities were usually absorbed. Various toxic substances, such as morphine, were injected, and it was found that they produced paralysis often localized to the point in which the injections were made, particularly in the lumbar region of the cord. An injection of microbes merely showed what an important part the cerebrospinal fluid played in their distribution. In the case of a patient suffering with tetanus, anti-toxin was injected into the lumbar region without any bad effects, and the patient remained alive for three days. Sicard calls attention to the recent employment of cocaine as a general anesthetic by injecting solutions of the drug into the subarachnoid space of the cord, and has repeated these experiments with success upon animals. SAILER.

72. BROMIPINA NELL' EPILESSIA (Bromipine in Epilepsy). P. Bodoni (Rivista di Patologia nervosa e mentale, 4, 1899, p. 390).

Bodoni reports on eight cases of epilepsy treated with this drug. By reason of its slow elimination and because it is irritating to the stomach, he believes it to be inferior to the bromides as usually prescribed, and that it is therefore not to be especially recommended.

JELLIFFE.

- 73 ON THE TREATMENT OF PAIN IN TABES DORSALIS; REPORT OF A CASE. Alfred H. Lindström (The Boston Med. and Surg. Journal, Jan. 11, 1900, p. 37).

The writer reports a case of tabes in which the pains were successfully treated by mechanical means alone. The patient, a man forty years of age, had pains for ten years, but the other tabetic symptoms did not become manifest until during the last year. He had been unwilling to take drugs for the pain, lest he should contract a drug habit. But the pains had been so severe that he finally resorted to whiskey freely during an attack.

There was no doubt of the diagnosis. His family and personal history were good—no syphilis or alcoholism. About thirteen years ago, however, he had fallen and injured his back slightly. At the time of beginning treatment the patient had been unable to sleep for ten days because of the severity of the pains. Still, he would take no drugs, although he was actually frantic with pain.

Lindström decided to try stretching of the sciatic by posture, namely, by flexion of the thigh and extension of the leg and foot. Each sciatic nerve was kept in a state of tension for two minutes at the first treatment. Numbness of the feet and slight distress in the popliteal space followed the treatment, but there was freedom from pain for several hours. On attempting to walk, the ataxia had apparently increased, but this was probably due to the numbness of the feet, since the gait improved as the numbness wore off. The patient was able to get several hours' sound sleep. The following day each sciatic was kept in tension for three minutes. The numbness of the feet and the apparent increase of ataxia immediately followed the treatment as before, but were perhaps more marked. The absence of pain was more complete. The patient slept well all night long. The next day a third treatment was given, two minutes to each sciatic. No pains having appeared, the next stretching was done two days later, and a fifth and last treatment forty-eight hours after that. During all this time the patient had been practically free from pain.

Since these treatments he has had only a few slight attacks of pain, usually caused by injudicious physical exercise. The general treatment of the case was tonic, eliminative, and hygienic, with the addition of systematic co-ordination exercises.

The hip-joint is extremely flexible in the early stages of tabes. Lindström was able, after flexing the thigh upon the abdomen, to bring the patient's leg and foot on the level with his face. Care must be taken in doing this not to use undue force, or any sudden pressure, for fear of fracturing one of the bones of the leg. Subluxation at the hip-joint must also be guarded against.

The writer advances several theories, by one of which, perhaps, the success of the postural treatment may be explained.

BONAR.

## Book Reviews.

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A GROSS AND MINUTE ANATOMY OF THE CENTRAL NERVOUS SYSTEM.  
By H. C. Gordinier, A.M., M.D., Professor of Physiology and of the Anatomy of the Nervous System in the Albany Medical College; Member of the American Neurological Association; with 48 full page plates and 213 other illustrations, many of which are printed in colors, a large number being from original sources. P. Blakiston's Son & Co., Philadelphia, 1899.

The reproach, that English literature has contained no adequate treatment of the anatomy of the nervous system, compared with the works of the Continent, by Van Gehuchten, Dejerine, Obersteiner, Koelliker, and Edingen, has been removed from our language by the appearance of two works, both of superior merit, within the closing months of the past year. Barker, "On the Nervous System," and the present work of Gordinier, both of which are masterpieces. In a way they cannot be compared, treating, as they do, their subject matter from quite diverse standpoints. Both are necessary to the alienist and neurologist.

The subject matter in the present volume is discussed in some fourteen chapters, being divided as follows: (1) The Histologic Elements of the Nervous System; (2) The Spinal Cord; (3) The Medulla Oblongata; (4) The Cerebellum or Encephalon; (5) The Region of the Mid-Brain; (6) Region of the Third Ventricle; (7) The Membranes of the Brain; (8) Fore-Brain or Prosencephalon; (9) Histology of the Cerebral Cortex, together with the Minute Anatomy of the Centrum Ovale; (10) General Anatomy of the Interior of the Cerebral Hemisphere; (11) The Blood Vessels of the Brain; (12) Cerebral Localization; (13) the Embryology of the Central Nervous System; (14) Technic of the Macroscopic and Microscopic Examination of the Brain and Spinal Cord.

In a sense Gordinier does not depart from the methods of presentation of the anatomy of the nervous system at present in vogue. For the general student we believe this to be a wise plan, and certainly the present volume will prove of service in the dissecting room and laboratory. There is very little that is original in the book, the standard text-books of the continent, already mentioned, having been freely consulted. The facts that are fairly well established are here given *ex cathedra*. But few references are given, and thus the book will prove of secondary service to the investigator or to the student of neurology interested in many of the modern problems of the minute structure of the nervous system. The author has made an honest attempt to place in the hands of the English student a comprehensive and accurate text-book, devoid of the many intricacies of modern thought and speculation. For the average man the work will appeal strongly, the facts that he can use are readily found; for the more advanced, or perhaps better, for the more exacting and technical student of neurology, the work, while giving a short cut to the treatises of France and Germany, is somewhat of a disappointment. The deficiencies in this respect, however, are made good by Barker, which author's work cannot serve the useful purposes, for the general practitioner and student, that are subserved by the volume in question.

JELLIFFE.

LEA'S SERIES OF POCKET TEXT-BOOKS. NERVOUS AND MENTAL DISEASES. A MANUAL FOR STUDENTS AND PRACTITIONERS. By Charles S. Potts, M.D., Instructor in Nervous Diseases, University of Pennsylvania; Assistant Neurologist to the University Hospital, Philadelphia; Consulting Physician to the Hospital for Insane, of Atlantic Co., N. J. Illustrated with eighty-eight engravings. Lea Brothers & Co., Philadelphia and New York.

This is a text-book which presents the subject of nervous and mental diseases within 437 pages. It is unnecessary to say that it is intended to convey to students and practitioners only the outlines of the subject, to which further knowledge must be added, derived from more comprehensive writings. In this volume the subject is treated in a very concise and equally clear manner. Four short chapters are given up to histology, general pathology, symptomatology, and therapeutics. The author believes in the neuron theory and has classified the system diseases according to the neurons affected. This method he believes to be the correct one, although it may not be strictly accurate. In considering the various diseases the main points are well brought out. Attention is given to the etiology and morbid anatomy as well as to symptoms, differential diagnosis, prognosis and treatment. The insanities and idiocy are considered in 42 pages, and the chief points clearly presented. As a manual and serving as an introduction to further study of nervous and mental diseases this book is to be recommended. It is well gotten up, with a number of very good illustrations, and is printed upon very good paper. BONAR.

KRANKHEITEN DER PERIPHERISCHEN NERVEN. Geh. Medicinalrath, Prof. Dr. A. Eulenburg. Separat Abdruck aus Handbuch der Praktischen Medicin, unter Redaktion von Dr. W. Ebstein und Dr. J. Schwalbe; herausgegeben von W. Ebstein. Verlag von Ferdinand Enke, Stuttgart.

This further chapter from the "Handbuch der praktischen Medicin," while betraying extensive personal experience on the part of the writer, has not been written without careful consultation of the literature. It begins with brief introductory sections on the anatomy, physiology and chemistry of nerves, and these are followed up by others which treat very satisfactorily of the technique of examination. In the main body of the work a just sense of proportion is maintained throughout, each nerve territory being given the prominence to which its importance properly entitles it. In the matter of treatment, the author shows himself resourceful, but presents no therapeutic measures which deserve particular mention. The illustrations are five in number and are good as far as they go. If any criticism is to be offered of the work, it is reference to this very paucity of illustration. To be absolutely adequate to the purpose for which it is designed, it should be accompanied by charts showing the motor points of nerve-trunks and the topographical distribution of cutaneous nerve territories.

J. W. COURTNEY.

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THE  
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**Nervous and Mental Disease.**

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**Original Articles.**

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A CASE OF WERNICKE'S CONDUCTION APHASIA WITH  
AUTOPSY.\*

By HOWELL T. PERSHING, M.D., of Denver, Colo.

Next to Broca the greatest credit for our present knowledge of aphasia is due to Wernicke.<sup>1</sup> In his monograph entitled "Der Aphasische Symptomencomplex," published in 1874, some time before the appearance of Hussmaul's work, he gave the first comprehensive sketch of the forms of aphasia, in which the importance of the auditory center for words was recognized for the first time and its location in the upper temporal convolution demonstrated. He clearly showed, from a consideration of the way in which speech is learned combined with a study of cases, that the utterance and especially the selection of words is normally guided by a memory of their sounds, and that this guidance is by means of association impulses from the auditory center to Broca's center; hence the paraphasia which is so conspicuous an accompaniment of the word deafness in cases of destruction of the auditory center. Taking a necessary step further, he concluded that a simple interruption of the path between the two centers must also prevent the auditory center from exercising its normal control over utterance, and he named the resulting defect conduction aphasia (*Leitungsaphasie*).

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\*Read at the twenty-sixth annual meeting of the American Neurological Association, May 1, 2 and 3, 1900.

<sup>1</sup>(Wernicke, "Gesammelte Aufsätze und kritische Referate zur Pathologie des Nervensystems," Berlin, 1893, pp. 25-27.)

The symptoms of conduction aphasia are made clear by comparison. He first states that lesion of the auditory center causes word-deafness; loss of imitative speech; paraphasia, in which correct and incorrect words are mingled; agraphia; paralexia on reading aloud and, in the unlearned, loss of comprehension of written or printed words. He then states that conduction aphasia is precisely like this form in its combination of loss of imitative speech, paraphasia, agraphia, paralexia on reading aloud, and loss of comprehension of print; but differs in the absence of word-deafness. In later years he was convinced that loss of comprehension of print or writing would be a necessary symptom in the learned as well as the unlearned. It was assumed that the association path from the auditory center to the motor-speech center was a direct one, through the insula, and "insular aphasia" is sometimes used as synonymous with conduction aphasia.

As a proof that conduction aphasia has a clinical existence, Wernicke published two cases in his original monograph. The first is that of an apothecary, aged 64, who, without any other disturbance, on waking one morning, found himself unable to read or write and, therefore, traveled to Breslau to see an oculist. Not till three days later did his speech become affected. Then his utterance was quite good until inability to recall a name embarrassed and excited him, when it became very much confused. When missing names were suggested to him they were recognized and repeated. Comprehension of what others said was good. On attempting to read aloud there was complete alexia, except for a few very familiar words and there was an entire loss of comprehension of print. He could not name single letters, but objects and pictures were recognized. Spontaneous writing was absolutely lost, but copying remained. There was right homonymous hemianopia. The diagnosis was of a lesion, beginning in the tuber cinereum, encroaching on the anterior perforated space and causing circulatory disturbances in the insula, thus interfering with communication between the auditory and motor-speech centers. The other case presented similar defects in the use of language, together with a probable right hemianopia, but could not be so well observed, as the patient was demented.

In the light of our present knowledge one can have but little doubt that the first of these cases was one of cortical visual aphasia, due to lesion of the angular gyrus, and, in justice to Wernicke, I hasten to add that he evidently has long since changed his mind as to the significance of both cases. Of the first he says in a footnote to his "*Gesammelte Arbeiten*," that he would now make a very different localization diagnosis. And in another place he says that, as these two cases were complicated, further observations must be awaited before coming to a decision as to the causes of the symptoms of conduction aphasia. Again he says that, as to the location of the lesions which find their functional expression in conduction aphasia only the scantiest and most uncertain material is at hand.

Of a case published by Lichtheim<sup>2</sup> as certainly a clinical example of conduction aphasia, Wernicke<sup>3</sup> remarks that he cannot so regard it, because comprehension of print remained good. This case may also be objected to because the autopsy showed the whole of the Sylvian fossa to be softened and both the third frontal and first temporal convolutions to be somewhat damaged, so the aphasia may be regarded as of a mixed form due to two incomplete lesions.

Notwithstanding the failure of the cases cited to establish this form of aphasia as a clinical and pathological reality, Wernicke seems to have steadfastly adhered to his original theory, confidently awaiting the evidence that would prove it correct. And, on any theory of aphasia which admits that the auditory and motor-speech centers are separate and connected by an association path, his reasoning must be regarded as sound.

It does not appear probable, however, that this association path is a direct one, through the insula. Three years ago, in a paper read before this Association, I<sup>4</sup> remarked that it is only

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<sup>2</sup>Lichtheim, *Deutsches Archiv f. klinische Medizin*, 1885, Vol. 36, p. 216.

<sup>3</sup>Wernicke, "*Gesammelte Arbeiten*," p. 121.

<sup>4</sup>Pershing, "Auditory Aphasia," *JOUR. NERV. AND MENT. DIS.*, Sept., 1897.

an assumption that this path passes directly forward—an assumption not justified by a study of lesions of the insula—that it much more probably takes a curved course around the posterior extremity of the fissure of Sylvius and that the *Leitungs-aphasie* of Wernicke is more likely to be found as a result of lesion of the supramarginal gyrus than of the insula. Recent anatomical authorities might be cited in favor of this opinion, and a case I have observed gives it clinical and post-mortem support.

T. E. P., a man of robust appearance, aged 45, was brought into the hospital in an unconscious condition, Nov. 19, 1897. The meager history obtained indicated that two days before, while driving, he became unconscious and fell from his wagon into the bed of a small stream, where he lay for about 36 hours before being found and removed to the hospital; also that on two previous occasions he had become unconscious, but had apparently fully recovered.

On examination he was found to be completely unconscious, the right arm and leg rigid and tremulous, the head and eyes showing slightly marked deviation to the right. There was no sign of injury to the head; pulse and heart were normal; respirations 20, somewhat stertorous; temperature 99.4-5; urine normal. A diagnosis was made of a vascular lesion, presumably syphilitic thrombosis, in the left hemisphere, and inunctions and potassium iodide were ordered.

In the course of a week consciousness had completely returned, but the man's talk was a mere jargon, which he shouted in a loud voice. There was also some word-deafness; he generally failed to do what he was told to do, but responded readily when shown by a gesture. No paralysis could be detected. During the second week in the hospital the word-deafness disappeared, and no sign of a cerebral lesion then remained except jargon paraphasia and certain associated defects. Seven weeks after the stroke his condition was noted as follows: Gait, station and the muscular actions generally are normal. On being told in a low tone, without gesture or accompanying change of expression, to perform certain acts he responds readily and accurately. He evidently understands the inquiries made of the nurse and interne as well as of himself, and is eager to answer. In doing so he uses "yes" and "no" accurately, but on attempting to express himself at any length, although the first few words are often intelligible, and apparently appropriate, he soon passes into a fluent but incomprehensible jargon, in which only here and there can a correct word be heard. Rarely the



whole of a conventional sentence will be correct and appropriate, as "Very well, I thank you." In trying to answer he never repeats any of the words of the question. He is entirely unable to name familiar objects.

On being asked to read aloud from a newspaper he says that he cannot, but he willingly tries. His reading is markedly paralexia: a few short words are correctly pronounced and the pronunciation of others bears an obvious resemblance to the correct sounds, but these are so mingled with unintelligible sounds that the whole conveys no meaning to a listener. He has no understanding of what he tries to read. The simplest directions given in writing elicit no response, although in studying them he utters some of the words correctly to himself.

He writes his own name quite legibly, only putting an extra "s" on the end. He is absolutely unable to write anything else, either spontaneously or at dictation, and, while the result of all his attempts is illegible, there often recurs a resemblance to parts of his name.

Acuity of vision is good. There is no hemianopia. He has a keen eye for all that goes on about him and often laughs at another patient whose appearance and actions are comical.

The patient's condition remained substantially the same in every respect until Jan. 23, when he suddenly began to vomit and was found to be unconscious. When I saw him next day there was rigidity of the left arm and leg, which were motionless, although the right limbs were moved in a jerky way as though he were resting uneasily. There was conjugate deviation of the head and eyes to the left, and the left pupil was larger than the right. He was quite unconscious. Pulse 64, weak and soft; heart sounds normal. Temperature in right axilla  $97.7^{\circ}$ , in the left  $97.2^{\circ}$ . Respiration of the Cheyne-Stokes character. Nails blue. Unable to swallow. Cardiac and respiratory stimulants caused temporary improvement, but death occurred from intercurrent pneumonia Jan. 29.

Autopsy 24 hours after death. Brain firm and well preserved. A general inspection of the membranes and larger vessels revealed nothing abnormal. Careful dissection showed nothing abnormal in the right cerebral hemisphere, but in the left there was a single spot of softening immediately above the fissure of Sylvius, 6 cm. back of the junction of its vertical and horizontal branches. This spot was in the form of an irregular cone, the base being an oval area at the surface, 1.8 cm. in vertical diameter, and 0.8 cm. wide. The long axis of the cone extended inward 2.5 cm. to about the same depth as the floor of the fissure of Sylvius. The pons and medulla

were normal, but the dorsal half of the right cerebellar hemisphere was completely destroyed by softening.

After hardening in formalin a more minute dissection of the left cerebral hemisphere was made, especially in the regions of the angular, first temporal and third frontal convolutions, but no additional lesion was discovered. All the arteries examined appeared normal to the naked eye, except one small one in the floor of the fissure of Sylvius near the spot of softening. At a point where this artery divided into a number of branches its wall was extensively and irregularly thickened, so that the lumen was almost obliterated.

Taking the clinical and post-mortem facts of this case together there can be no doubt that the aphasia was caused by the lesion in the left cerebral hemisphere. Motor aphasia was excluded by the large number of words correctly uttered. Visual aphasia was excluded by the absence of hemianopia and the considerable number of words read aloud, either correctly or incorrectly. The defect was clearly a form of auditory aphasia, but was differentiated from other forms by the rapid disappearance of the word-deafness without any change in the paraphasia, paralexia and agraphia. The lesion was small and well defined, and according to the latest anatomical authorities was so situated as to interrupt communication between the first temporal and third frontal convolutions.

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- 74 TUMEUR CONGÉNITALE DE LA RÉGION LOMBAIRE (Congenital Tumor of the Lumbar Region). P. Piollet (*Nouvelle Iconographie de la Salpêtrière*, January and February, 1900, 13th Year, No. 1, p. 71).

A congenital tumor of the lumbar region, which does not belong to the class of lipomata found in this region nor is it a variety of spina bifida. The tumor is flat, pediculated, is suspended from the median line at the lower dorsal region, about the size of a hand and the thickness of a finger, and is congenital. When the child began to walk, the tumor gradually increased in size until its weight became a serious embarrassment to locomotion. A quantity of serous fluid exuded from the skin covering the tumor. The skin over the tumor is pigmented. The tumor was removed by operation; it weighed about six kilos. Pathologic examination showed connective tissue formation, malignant in nature by reason of a mucoid tissue. Malignancy purely local, susceptible of local recurrence, but not of generalized growth. It is probably a congenital fibromyxoma of the lumbar region, originating from the posterior vertebral arch. It is similar to a solitary molluscum, but differs from it by reason of its malignant evolution. SCHWAB.

## A CLINICAL STUDY OF SOME REFLEXES.\*

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AND

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This inquiry into the nature and significance of some of the more important reflexes is based upon upward of three thousand recorded examinations made upon healthy and sick persons in the Montefiore Home, and in our clinic for nervous diseases at the Post-Graduate Medical School. About two-thirds of the entire number of persons investigated were sufferers with nervous diseases.

The first object of the study was to determine, if possible, the physiological nature of those reflexes called tendon phenomena, and to inquire into their relationship to, and probable dependence upon, muscle tone. Another object was to ascertain the behavior of tendon jerks under repeated stimulation by a blow sufficient to elicit the jerk; in other words, to determine the exhaustibility of the tendon reflex. Finally the relation between deep and superficial reflexes was studied, and the claim recently put forward in regard to the semeiological significance of the plantar reflex was investigated.

The examinations were made in the following order: Two hundred and ninety individuals were examined with a tonometer, which measures the tonicity of the Achilles tendon. This instrument was devised by Dr. L. J. J. Muskens, a former assistant in the clinic, who published a description of it in the *Neurologisches Centralblatt*, December 1, 1899.

With this instrument sixteen measurements were made on each foot; that is, thirty-two on each patient, then the average mean of this number was taken to represent approximately the tone of the muscles.

The following topics were selected for particular investigation:

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\*Abstract of a paper read before the American Neurological Association, May 1, 1900.

1. The state of tension of the patellar tendon, determined empirically by palpation.

2. The knee-jerk. The blow to elicit this was struck first with the hand and then with the hammer, while the knees were crossed, uncrossed, and with and without reinforcement, each test being made ten times in succession.

3. The Achilles jerk. This was examined in the following manner: The foot was placed on the floor in such a position that the examiner had sufficient space to swing the hammer in order to give the tendon an effectual blow, while the position of the examiner was such that he could see the quadriceps muscle contract, and at the same time observe the amount of locomotion produced by the contraction. In this way both phenomena, the contraction of the muscle as well as the locomotion of the foot, were seen and studied, while the speed of the other reactions were also observed.

4. The plantar reflexes were selected for most extensive observation, not only because they are the most constant of the skin reflexes, but because they fairly represent the type of such reflexes. In the first group of examinations the object was to ascertain the inter-relationship of tendon and skin reflexes. Another series of examinations was made for the purpose of ascertaining the speed of the tonus reflex or the reaction time, particularly that part of the tonus constituting the reflex act.

5. Finally, in every patient the mechanical irritability of the quadriceps muscle, as elicited by percussion, as well as the speed and quality of this reaction was determined.

The first matter for consideration is the relationship between the tendon reflex and muscle tone as determined by the tonometer. As a preliminary observation it is necessary to say that the question of the existence of muscle tone and its influence upon the tendon reflex is a mooted question with both clinicists and physiologists. It may be objected by some that our statements concerning muscle tone are based on examinations of the Achilles tendon, and that the result of these findings are applied to the state of the patellar reflex. In response to this we say that there is no valid objection to the assumption that the results of these measurements may not be taken as a reliable index of general muscle tonicity.

The tonometric figures in all the cases may be divided into three groups: First, Those ranging from 17 to 27, which are referred to as cases with large tonometric figures; second, those from 12 to 17, medium tonometric figures; and third, those below 12, as small tonometric figures. Those with figures from 12 to 17 may be looked upon as representing the average of physiological tonus. All figures above this may be called evidences of hypotonus, while the figures below it may be considered indicative of hypertonus.

The first point for consideration was to determine if there was any exaggeration of knee-jerks, with large tonometric figures. Of the cases with large tonometric figures the knee-jerk was absent in 16 cases: 14 of tabes, one of Friedreich's ataxia, and one of peripheral neuritis. It was diminished in 15 cases. We speak of it as diminished when the Jendrassik method had to be employed or the knee-jerk had no locomotion. In twenty cases it was lively or normal.

The highest tonometric figures in the group with high figures are found in those in which the reflexes were absent, but not in all. This rather surprising finding is explained by the fact that those patients were confined for a long time to a chair or bed, and that the tendon was soft, atrophied, and close to the bone, so that the rod of the tonometer was pressed against bone, and not against tendon.

The group of cases with diminished reflexes represents all kinds of internal diseases. It is made up mostly of scrofulous and phthisical patients, the latter with evidences of amyloid degeneration, and those with functional nervous diseases, neurasthenia and hysteria of the gastro-intestinal variety.

After finishing with this large group, we must consider the hypertonic group. We find a very small number, viz., 22, with absent or diminished reflexes, the remainder showing either normal or exaggerated reflexes. Eighteen show increase of knee-jerks and ankle-clonus. From the group that showed exaggerated reflexes we select first, those that not only had exaggerated knee-jerks, but ankle-clonus as well, 18 in number. They all represent cases of diseases of the pyramidal tracts, one sided or both sided, with one exception, which was a case of paralysis agitans. So that great value is attached

to them, particularly those in which the disease was one sided, as well as in those in which there is a very distinct difference between the diseased side and the healthy side, in that the side upon which the disease existed showed at the same time smaller tonometric figures.

It now becomes necessary to explain the twenty-two cases with diminished or absent reflexes. The first point that attracts our attention is that this group contains a number of cases of *tabes*, viz.: 7. The majority of the patients making up the group with diminished reflex, however, were muscular, robust and healthy individuals. One case was that of a patient with *paramyoclonia*, a disease characterized by a state of exalted muscle tone. In another case there was a cardiac lesion, with incompetency, and so much edema of both the knee and the ankle that neither tendon could be reached so as to apply the tonometer. In another case the same difficulties arose from edema produced by liver disease. In still another case, with disease of the pyramidal tract, our notes state that the tonometric figures and the behavior of the reflex could not be got nor a reliable examination made, owing to the incessant tremor or clonus. In still another case the patient could not be made by any means to relax the tendon. A similar statement applies to other neurasthenic and excitable patients, and so there is finally left only two cases to explain; one with an aortic insufficiency, in which no apparent reason could be given why the reflexes were diminished; and another extremely interesting case, a case of a tumor of the left cerebellar hemisphere, found by autopsy, in which the measurements were taken in bed on various occasions, and were found to differ on every examination. At one time the hypotonus would be excessive, and the reflex not elicitable, and at another time the tonus increased and the reflex lively, the ankle-clonus being easily elicitable.

In the group with small tonometric figures there were a number of patients with lively or slightly diminished reflexes, which call for no further explanation. In this group, with tonometric figures of from 12 to 17, we find either lively or somewhat lessened reflexes, depending upon whether the figures are nearer to the upper or to the lower limit.

In general and in brief, it may be stated that with increased

muscle tone there is increase of tendon reflexes up to a point of pseudo-clonus, and with diminution of muscle tone there is a diminution of the tendon jerk up to a point of apparent disappearance. When the increase of the reflex has progressed so far as to give rise to a genuine ankle-clonus, or decreased so far as to lead to total abolition of the reflexes, there are other and additional factors than increase or diminution of muscle tone to be found in explanation of it: such as purely neural factors, like disease of the pyramidal tracts or disease of the posterior columns.

From the foregoing, the inference seems justified that tendon reflexes are not elementary, but complex phenomena. Their existence is dependent upon at least two, perhaps only two, factors. First, the state of nutrition and the reaction of the muscle to proper stimulus, and secondly, the state of the reflex arc carrying and bringing the nerve supply to the muscle. That this is true is very evident when our tables are consulted, which show the relationship between tonus reflex and mechanical muscular irritability. It was found that whenever the diminution of the reflex was due to a disease of the neural elements the muscular irritability was found to be increased; when, on the other hand, it is due to an interference with those factors themselves—the state of nutrition and tonus of the muscle—the mechanical muscular irritability was diminished, vermicular and sluggish. This is particularly evident in the cases of tabes, in which our examinations showed, without exception, the curious phenomena that the quadriceps muscle struck at its tendon gives no response, while the same blow applied to the belly of the muscle is followed by quick and vigorous contraction.

Empirically, by means of percussing the patellar or Achilles tendon an approximate estimate of the state of tension of a given tendon can be made, although not so exact as with the instrument. When action is exerted during the examination and the patellar tendon examined while the patient's foot rests on the floor, the leg forming a right angle with the thigh, the patient thus reclining comfortably, while his attention is diverted, it will be found that in tabetic and otherwise hypotonic patients the tendon is remarkably soft and easily compressed.

The plantar jerk is a typical representative of the skin reflex, as well as the most constant one. We have never found it absent without adequate attributable cause, such as disease of the peripheral nerves. In profound neurasthenia this reflex is considerably diminished, it is true, but it is never absent. In examining this reflex it is well to make a difference between strong and mild stimulation, and between the stroking and the sticking reflex. In the first group of cases examined attention was paid only to its presence or absence, and no particular notice was taken of the succession of events constituting the reflex phenomena. At this time it was found, as has already been observed by others, that the tendon reflex and the skin reflex show a peculiar dissociation. With increase of the tendon reflex there is usually a diminution or abolition of the skin reflex, but the reverse of this is not so unconditionally true. It was further remarked that this reflex is to a great extent dependent upon the state of the susceptibility of the part irritated. This is abundantly illustrated in cases of tabes. Here the plantar reflex is frequently delayed or duplicated, and one ought, therefore, to wait at least one minute before he pronounces that the plantar reflex is absent. A further interesting clinical phenomenon that is helpful in concluding as to the nature and location of a spinal lesion is the fact that total anesthesia and total motor paralysis do not involve the plantar reflex, so that the conduction to the particular anterior horn cells by means of the reflex medullary arc is not disturbed, while conduction of the same stimulus to the sensory centers is interfered with. And so it is explained that this reflex is the only evidence of the reflex activity left after total transverse section of the cord.

After attention was attracted to the symptomatic importance of the reflex observed by Babinski, a large number of examinations was made to determine the succession of events constituting it, *i. e.*, the "Babinski reflex," and from our observations the following seems to be the physiological formula: After mild stimulation of the plantar surface in normal individuals the first response is a slight contraction of the quadriceps imparted through and determined by contraction of the tensor fascia lata. The next incident is a flexion of the four smaller



toes, with plantar flexion of the foot and dorsi-flexion of the big toe, slight flexion of the knee and hip, and abduction of the latter. The phenomenon that has been so far of the greatest clinical importance is the response of the toes. The physiological condition of this response consists of a more or less vigorous plantar flexion of the four smaller toes. When the reflexes are exaggerated, all toes, including the big one, flex. In disease of the pyramidal tracts this formula is reversed, and in varying degree. There may, in mild lesions, be only a slight transitory flexion of all the toes, and in well-defined cases of disease of the pyramidal tracts the first response noticed is a transitory dorso-flexion of the big toe. This, manifestly, is the cause of the hyperextension of the toes so frequently found in spastic states. From our observations we have corroborated the findings of Babinski, van Gehuchten, Collier, and others, that this type of plantar reflex is not found in normal individuals, and that when carefully examined with the precautions mentioned above, it is found only in cases in which there is unmistakable evidences of disease of the pyramidal tracts.

Finally, the writers desire to say a word anent the customary ways of examining the knee reflex. With most observers, the examination is undertaken simply to ascertain its presence or absence, while no particular note is taken of the quality of the response or of the behavior of the reflex after repeated elicitation. Bearing this in mind, we feel justified in stating that in a number of instances this reflex shows a peculiarly whimsical and interrupted behavior after repeated examinations. When a number of tapplings are made in succession, one will find that the responses vary in intensity in a considerable degree, or that here and there one reflex is missing, and before that it may have been suppressed. If one is attentive at the same time to the palpitatory impression received from the tendon, he will find that this whimsical reflex often coincides with a very soft tendon.

#### DISCUSSION.\*

Dr. S. Weir Mitchell said he had listened with interest to this paper, and it was on a subject to which he had given much

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\*This discussion refers also to the paper by Drs. Walton and Paul, p. 305. *et seq.*

thought. After Jendrassik discovered the simple fact that to shut the hand firmly increased the knee-jerk, he dropped the subject. It was taken up by Dr. Mitchell, and, with Dr. Morris Lewis, carefully studied. They showed that all muscle acts reinforce the knee-jerk. They discovered that all forms of sensory excitement do the same—and much else which no foreign text-books remember to use. Lombard followed with laboratory confirmation and brilliant additions. Bowditch discovered the secondary inhibition, or lessening causes. The studies by Drs. Mitchell and Lewis of muscle-jerk, etc., and other American papers, followed. In fact this whole study has been the work of the two former observers, and the results long ago made clear are being continually rediscovered.

Dr. Collins had spoken of muscular tone. It is really a double contribution, as Drs. Mitchell and Lewis have proved. First there is the inherent muscle quality which causes a muscle to move when struck. This is still seen in muscle isolated from the centers. In muscle normally connected with the spine there is also a contributory tension of muscle due to central influence, for if a blow on muscle causes it to move, all agencies which reinforce knee-jerk add vigor to muscle-jerk. But the knee-jerk due to a pull on the muscle, requires also some mechanical tension to be effective and is so much more delicate than direct muscle-jerk as to be easily lost; but the phenomena are alike in kind.

There are many unsolved problems; the blow of a hammer on tendon or muscle causes movement capable of being reinforced by remote movement or sensation. But electrically excited motion of muscle is not thus capable. Why, is not known. Again, if knee-jerk be a reflex act, as it seems to be, and reinforcing, why are not the skin-muscle reflexes susceptible of such additions? Again, in some spinal diseases you can reinforce enfeebled knee-jerks by motion at a distance and not by sensation, or the reverse of this may be true. What, then, are the tracts of reinforcement?

Again, how can we explain the absence of these reflexes in some normal people? In them, as no one has yet pointed out, the muscle-jerk exists, but is not reinforcing; in other words, is not fed with spinal tension tone.

Dr. Mitchell concluded by saying that he feared that in place of helping he had simply increased the difficulties which surround this subject, but that when you increased difficulty you increased interest, and with a body like this, this was the right thing to do.

Dr. P. C. Knapp said that there were two other peculiarities about the skin reflexes which Dr. Mitchell might have

mentioned. The first is, why are they so persistent in tabes; the second, why, with the exaggeration of muscle-muscle reflexes in hemiplegia and spastic conditions, the skin reflexes are often diminished.

The question of plantar reflexes, which had been dwelt upon by Dr. Collins and Dr. Walton, was one which had interested Dr. Knapp particularly of late, and he was much more disposed to agree with Dr. Collins, in believing that the plantar reflex was almost always present in health, than he was with Dr. Walton. He had not tested it very often in healthy people, but certainly in testing it in people who are sick, he had found hardly a case where the plantar reflex was absent unless there were some pretty definite reasons therefor, due to some change in the nervous condition.

Dr. Collins had hinted at a partial explanation of this. In some cases the stroking stimulus is insufficient to produce it, it requires the sticking stimulus or the summation of irritation, the irritation of a long-continued stimulus. In other cases there are not special movements of the toes, but we find a fascia lata reflex, which is a little more difficult to observe, since the patient must be completely stripped. In other cases still we may observe a purposive movement of withdrawal, which Dr. Knapp had found without apparent clinical significance in a number of cases, where there was not the normal flexion of the toes. In a few instances, especially in cases of chorea and in neurasthenic and hysterical conditions, he had found extension of the small toes without extension of the great toe.

There are two or three points with regard to the Babinski phenomenon pure and simple, which are worthy of mention. In the majority of cases, of course, it seems an indication of trouble in the pyramidal tract. Dr. Knapp recalled one case showing the Babinski phenomenon, where there was at first a diagnosis of tabes, but which proved to be double sciatica with muscular degeneration and paralysis of the extensor of the foot. In that case stimulation of the sole caused the Babinski phenomenon. He had found other cases with exaggeration of the deep reflexes, such as amyotropic lateral sclerosis, where there was no Babinski reflex, but a normal flexion. In regard to the period of time in which the Babinski phenomenon may develop he had seen a few weeks ago a case of extra-dural hemorrhage, with an enormous hemorrhage pressing on the brain, the clot being at least an inch and a half thick. He had seen the man four hours after the accident and three hours after the onset of the coma and hemiplegia. At that time there was a Babinski reflex in the paralyzed leg. Dr. Lothrop

trephined and removed the clot about nine o'clock that evening, and the next morning the man had recovered completely from the paralysis and there was the normal flexion reflex.

A second case, however, was more important, although he could not explain it. An old hemiplegic with contracture had a Babinski reflex in the paralyzed leg, and the normal flexion reflex in the unparalyzed leg; but on stimulating the unparalyzed leg it produced normal flexion in the great toe of that leg and also normal flexion in the paralyzed leg.

Dr. Knapp could not agree with Dr. Walton that the plantar reflex is often unlike on the two sides. He had not found such a difference unless there was some disease of the nervous system.

Dr. B. Sachs said that it was a curious fact how often this question of reflexes is gone over again and again; we have been treated to the same facts that we heard ten or twelve years ago, with the exception of the Babinski phenomenon.

He wished to recall that twelve years ago he wrote to Dr. Mitchell about a case in which the tonus of the muscle was remarkably preserved, while the knee-jerk had disappeared, and Dr. Mitchell kindly sent him in return some "curves," showing that muscle tonus and deep-reflex were entirely independent of each other. He did not know whether Dr. Mitchell still holds to this view. As for himself he could not help feeling that hypotonus and lost reflex are coincident phenomena depending upon a lesion either in the spinal cord or in the peripheral nerve. He had this to add with reference to the Babinski phenomenon. He had observed it carefully in children and thought that it was a most unreliable feature in children. He had not been able to get any satisfaction in children up to years of nine and ten. He regarded the reflex as useful, but a very careful study will have to be made before we can give it the same value that we now attach to the knee-jerk and plantar reflexes.

Dr. W. G. Spiller referred to some observations he had recently made in two cases of hemiplegia. One patient was seen about one week after the hemiplegia developed. In each case the knee-jerk was absent, and the Babinski reflex was very pronounced; and in one case, at least, the knee-jerk did not return two or three weeks after the hemiplegia developed. Both patients died. Two cases are not sufficient to draw conclusions from, but Dr. Spiller believed that in recent cases of hemiplegia in which the knee-jerk was found absent several days or weeks after the hemiplegia had developed and the Babinski reflex was present prognosis was bad. It would be desirable if attention were paid to this combination of symptoms.

He had seen a case of amyotrophic lateral sclerosis in which the atrophy of the hand muscles was very marked, the knee-jerks were much exaggerated and the Babinski reflex was not present. In another case many symptoms of tabes were present, but the knee-jerks were exaggerated and the Babinski reflex was absent. This was probably a case of spinal syphilis. In a case of fracture of the spinal vertebræ with paraplegia, Dr. Spiller had observed extension of the great toe of one side with flexion of the other toes of the same foot. The Babinski reflex he regarded as of considerable value in connection with other signs, but it was doubtful whether it could be looked upon as pathognomonic of lesion of the pyramidal tract.

Dr. F. W. Langdon said that in his experience the plantar reflex had been actually absent with extreme rarity, possibly not more than in one or two cases in one hundred; and in cases where it had been *apparently* absent, it had been proven to be present subsequently by carefully testing the patient, without letting him know when it was to be done. Therefore, when Dr. Langdon intended to make a test in a doubtful case he would direct the interne or nurse to leave the patient uncovered and blindfolded, and the test would be made unexpectedly to the patient. Dr. Langdon had found Babinski's phenomenon an exceedingly reliable clinical sign, and it had been of service to him in a number of cases where the knee-jerk was of very doubtful value. He considered it, therefore, a sign of considerable clinical importance.

TUMOR OF THE PARIETAL CONVOLUTION, ACCURATELY LOCALIZED AND REMOVED BY OPERATION.

By C. K. MILLS, M.D., and W. W. KEEN, M.D.

(See p. 244).

DISCUSSION.

Dr. F. X. Dercum said that this, to his mind, was one of the most instructive cases that he had ever seen. Too much credit cannot be given to Dr. Mills for the brilliant and successful outcome of the case. Dr. Dercum saw the patient in 1898, two years before the operation. He was then simply somewhat depressed, but outside of an accentuated second sound of the heart, he presented not a single physical sign. He had, however, one subjective symptom, namely, an occasional mouse-like sensation starting from the head and extending to the arm. The symptom of astereognosis was of very great value in localization. Dr. Dercum was quite certain that the symptom of astereognosis is not as much studied as it should be. He was surprised to find in his wards at Bockley, in going over 114 miscellaneous cases, thirty-one that presented the symptoms of astereognosis. It seemed also to be possible to determine in

most cases, when it was present, whether it was cortical or peripheral in origin.

Dr. G. L. Walton said it was of interest to place on record all such cases of established tumor, in the absence of headache, vomiting and optic neuritis. Though this possibility is recognized, it requires courage in the given case to make a confident diagnosis, and to act on the conviction. He had recently seen a case illustrating this difficulty. An elderly man had twitching in the flexors of the left wrist, followed by paralysis, then twitching in the extensors, followed by paralysis gradually involving the whole arm and leg without further spasm. The trouble came on six months ago, but there has been no headache, vomiting, or optic neuritis. Dr. Keen and Dr. Peterson were consulted by letter, and on the same data the former diagnosed tumor, and favored operation; the latter regarded the case as one of thrombosis, and deemed operation unjustifiable. Dr. Richardson and Dr. Walton favored the diagnosis of tumor, and favored exploratory operation, but felt no confidence in the diagnosis, or prospect of relief. During the past week chills, restlessness and fever, and optic neuritis had appeared. In view of the evidently terminal nature of the present symptoms all idea of operation has been abandoned, but the optic neuritis favors the diagnosis of tumor, and probably a diffuse glioma.

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75. THE DEVELOPMENT OF VOLUNTARY MOVEMENT. E. A. Kirkpatrick (*Psychological Review*, 6, 1899, p. 275).

This is a brief discussion of an interesting topic in human genetic psychology, a topic still much in need of study based on observation.

The three theories of the attainment of the power of motor control are mentioned, the first, popular in its status, that the child learns how to make movements; the second, that the capability of making these movements is inherited just as in the case of the chick, and the third, "that the movements are partially provided for by the inherited mechanism and partly acquired and learned"; of these the author favors the last, while admitting that the second has many facts in its favor.

There is obviously, Dr. Kirkpatrick thinks, "an inherited physiological space-relation between the visual stimulus of an object in a certain position and the muscles for moving to that object." This physiological space-relation is deemed to be of an hitherto unappreciated importance in explaining not only the development of voluntary movements, but spatial ideas as well. In the first graspings and reachings, visual sensations alone are employed, but later on the joint and muscle and tactile sensations already experienced are helpful and important to further advance in the coördination of movements. "Chance" is shown to be of insignificant effect because of the mechanical complexity of the neuro-muscular organism. The child does not properly try to combine the elements of a movement and (as is the case with later and adult actions) does the movement more easily and better when least in the deliberate consciousness. The advantages of allowing free scope to the inherited spontaneity of action have important application in all forms of manual training and in active educational processes in general. DEARBORN.

## A DIGEST OF RECENT WORK ON EPILEPSY.

By L. PIERCE CLARK, M.D.,

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*(Continued from page 348.)*

### STATUS EPILEPTICUS.

The status is fairly well recognized now to be the acme or climax of the disease; that is, given the greatest number of contributing causes, the epileptic is foredoomed to die of status condition as the maximum development of the disease. Certainly chance plays no part in its production. Throughout all the present day definitions of status epilepticus, we find that the constant factors of rapidly repeated seizures and the progressive deepening coma are always included as the essentials. There is a marked elevation in the fever pulse, and respiratory curves. Some still hold that status is seen only in certain kinds of epilepsy, but by far the greater number of authorities maintain the more logical ground that the condition may occur in all epilepsies. An interesting point is disclosed by recent investigations, and there appears to be a necessity for reviving the old theory of cortical heat centers (a theory put forward by Eulenburg and Landois), to explain the high temperature in status epilepticus composed entirely of psychic attacks. The fever curve of status still remains mysterious, although it is generally in direct ratio with the severity and number of epileptic convulsions. Inasmuch as this rule is not always true, there are evidently latent factors in heat production in the epileptic paroxysms, which are not referable to the muscular convulsions and of which we have no present knowledge. The understanding of the anomalous fever curves in status will probably rest for ultimate solution upon its etiological pathology. After all, the enigma of the fever curve is not more difficult to unravel than the loss of consciousness in isolated convulsions of epilepsy proper.

The length of time after the disease of epilepsy begins and the time when status will occur is a point of great practical importance to all physicians. Recent statistics seem to prove that epilepsies developed in later life have status in a shorter period of time than when the epilepsy is contracted in early life. How-

ever this may be, it is certain that the disease of epilepsy in the majority of cases must be very well established before status will supervene. Epileptics may never have more than one status period, or may have six or seven, although the latter number is quite rare. Cases are not unknown in which patients have recovered from status and also from the epilepsy proper; but this result is very rare indeed.

The influence of menstruation and pregnancies upon the production of status in epileptic women is nil. It is as infrequent a clinical experience to see true status develop from the menstrual period as it is common to see serial attacks of major hysteria at such epochs. This fact alone aids one much in the differential diagnosis between serial attacks of hysterio-epilepsy and status epilepticus. The immediate cause of status is still as indefinite as that of epilepsy proper, and the solution of the problem rests upon the discovery of the pathology of the affection and the modifying agencies of the individual's resistance to the progress of the epilepsy. Its pathology beyond a vascular and cellular degeneration of the cortex is still obscure. Many writers have recently put forward different specific auto-toxic theories for explanation of the status, but none are generally accepted or even credited by the majority of neurologists. Oftentimes the vascular and cellular degeneration are really a result rather than a cause of the status. The actual onset of status does not differ from the beginning of serial attacks, and serial periods without status are frequently termed aborted or pseudo-status. Careful attention to these periods frequently delays the presence of true status epilepticus in the individual patient. The gradual, step-like advance of the grave symptoms of exhaustion and waste of energy is the cardinal factor in forming a prognosis of status epilepticus.

The advance of our knowledge of treatment is not great. A significant fact of the futility of medicinal treatment is seen in that all plans of medication are of service in isolated cases. The percentage of deaths remains about sixty or seventy in spite of improved methods of treatment. In the medication of status it should always be borne in mind that that which is indicated for the convulsive stage is contra-indicated for the stuporous stage; and that the administration of large doses of chloral, un-



combined with other drugs, has come to be considered as dangerous medication, in the light of depressing effects obtained from chloral upon weak hearts. Notwithstanding the frequent abscess formations from hypodermic bromide medication, this line of treatment is steadily growing in favor in the severest cases of status. The use of anesthetics still holds a deservedly prominent place. Good general nursing is nowhere more needed than in giving proper care to epileptics suffering from status.

DIAGNOSIS AND MENTAL STATES IN EPILEPSY.

Karplus has recently overthrown the differential diagnostic point of immobility of the pupil in epilepsy. In a study of 100 cases of hysteria major, he found rigidity of pupil in many of the latter affection.

Aside from the admirable work of Anton Deltil upon the medico-legal aspect of epilepsy, which is too exhaustive for careful consideration here, not many articles of importance have appeared in this section.

In regard to the post-epileptic mental states, Krafft-Ebing has contributed a valuable article upon the psychoses of epileptics. He found religious delusions interwoven with those of terror in the thirty-eight cases of post-epileptic delirium. It has been well known for a long time that epileptics are apparently predisposed to religious hallucinations; three epileptics were founders of religions: Mahomet, Swedenborg and Ann Lee, who organized the Shakers. Epilepsy and religious bigotry seem to be allied many times, and Krafft-Ebing is inclined to regard temporary religious frenzy as a species of typical epilepsy.

Marchand shows the rise of temperature after an epileptic attack varied from  $1.2^{\circ}$  C. to  $0.1^{\circ}$  C.; the mean rise was  $0.5^{\circ}$  C. The temperature returned to the normal in a quarter of an hour in 23 out of 102 cases; in half an hour in 9; and in from an hour to an hour and a half in 43; in 11 it had not begun to descend an hour after the fit; in 16 it was still rising at the end of the hour; in 80 out of 158 cases it had reached its maximum ten minutes after the onset. The temperature remained at its highest level for from 1 to 30 minutes, the average being 10 minutes. No relation

was observed between extent of the rise and the cause of the attack, nor between the former and the age of the patient. In the same patient the rise was found to vary both in extent and duration in different attacks. The rate of the pulse was increased on an average by 31 beats per minute. The mean quickening reached its maximum 16 minutes after the onset of the attack, remained there 5 minutes and returned regularly to the normal level in the course of 50 minutes. The relative increase in the pulse rate greatly exceeded the rise of temperature. Either pulse or temperature may be the first to reach the normal level. In epileptic vertigo there was a mean rise of  $0.3^{\circ}$  C. lasting on an average 40 minutes, reaching its maximum in 13 minutes, and returning regularly to the normal. The rise was of smaller extent than in the convulsive fits. The average increase in the rate of the pulse was 19 beats per minute, lasting 42 minutes, and maintained at its maximum rate for only one or two minutes. The rate may reach its highest point at the moment of the attack. The relative increase in the pulse rate was again much greater than the rise of temperature. This study of Marchand's throws much light on the differential diagnosis of epilepsy and hysteria; the latter disease has no pulse and temperature changes.

#### PROGNOSIS IN EPILEPSY.

In regard to the prognosis of epilepsy various authors place the recovery rate from 1, 2, 3, even 7 per cent. Le Duigon collected the cases of ten children who recovered at the famous schools at Bicêtre. Neurotic family factors were prominent in nearly all the cases. Nearly all the bad prognostic elements were represented; no laws governing the recovery could be ascertained. The lesson taught is that, no matter how bad the epilepsy, almost any case has possibility of recovery.

That epileptics are shorter lived as a class than normal people is well known, and accidents of various kinds resulting from paroxysms are subjects of frequent report. Féré has recently reported hernia of muscle in a single case, which resulted from violence of the muscular paroxysm. Some fifteen more were found with fascial openings thought to be congenital defects.

As to the cause of death in epileptics, Dr. Ballard gives an

analysis of the death of 260 fatal cases collected from Bourneville, at Bicêtre; the table is as follows:

|                            |    |                          |    |
|----------------------------|----|--------------------------|----|
| Status epilepticus .....   | 78 | Broncho-pneumonia .....  | 16 |
| Death in an attack.....    | 11 | Pneumonia .....          | 16 |
| Asphyxia from foreign body | 5  | Meningitis .....         | 2  |
| Asphyxia by hanging.....   | 1  | Cerebral hemorrhage..... | 3  |
| Asphyxia by drowning.....  | 1  | Sudden death (cerebral   |    |
| Cachexia .....             | 31 | tumor) .....             | 7  |
| Pulmonary tuberculosis.... | 41 | Fracture of skull.....   | 4  |
| Congestion .....           | 21 | Various disorders .....  | 23 |

As to what constitutes a cure in epilepsy much difference of opinion still exists. It matters little whether a cessation of attacks is called a remission or a cure. To many a cessation of paroxysms for two or four years is equivalent to a cure, and to others renewal of the disease, which is by no means infrequently the case, is termed by them a new attack of epilepsy. Still others hold that the renewal of paroxysms, no matter if they have ceased for twenty years, marks the return of the same disease, which has only suffered a remission.

#### TREATMENT OF EPILEPSY.

The treatment of epilepsy by bromide medication still holds first place in drug treatment, although many are yearly declaring against its use. Its abuse has been great, and the routine treatment of all cases by bromides is not only poor therapy, but actual, culpable negligence. Some cases tolerate bromides very well, others not at all. Laws governing one in the selection of cases where it will do no harm are still undiscovered. While large amounts of bromides are commonly borne with impunity, all drugs are frequently of no use even as adjuvants to hygienic methods. Gelineau has carefully considered such cases and aptly terms them cases of "intangible epilepsy"; intangible not only to bromides but to all treatment. Undoubtedly such cases have a gross organic lesion impossible of amelioration, and are fortunately not very common.

In order to avoid the necessity of giving large doses of bromides and thus cause an intolerance of the drug, Toulouse and Richet have devised a diet in which they endeavor to cut down the quantity of common salt which their patients take in food, the rationale of the treatment being that the less of sodium chloride there is in the economy the less bromide salt

necessary, as sodium chloride counteracts the effect of the bromide salt. Some thirty epileptics were treated successfully by this method; but one-half the usual amount of bromides (2 gm.) was used, while the daily consumption of salt was reduced from 14 gm. to 2 gm. The experiment was conducted carefully and seems destined to be of signal value in bromide medication in selected cases. Féré has very recently proven that if bromides are given to elderly epileptics with great care as to diet and hygienic states of the skin, etc., good results may be obtained, which is contrary to generally accepted belief that there is a marked intolerance of bromides in the aged.

The use of ipecac has been tried with bromides when treatment resulted in failure by use of bromides alone. It is believed that the ipecac counteracts the boulimia and resulting bolting of food so common in certain cases of epilepsy. The opium bromide treatment, which never had much to recommend it, has not been favorably commented upon in recent reports.

J. G. Smith, in a very careful résumé on the comparative use of strontium bromide and potassium bromide, concludes that potassium bromide will not be superseded for the four excellent reasons: Rapidity of action, durability of effect, smallness of dose, cheapness. Wright's study of the brain of an epileptic, who died of enormous doses of bromide, proved to him that the drug attacks the peripheral protoplasmic processes of the ganglion cells of the cortex. Von Bechterew reaffirms his previous statement in regard to the good results to be obtained in giving *adonis vernalis* with bromides, especially when there are cardiac symptoms indicating its use. One formula given by him contains potass. brom., sod. brom., codeia and infus. digitalis.

Trional has been tried by Maunier, who gives it in the doses of gr. v to xx with good effects; results contrary to those obtained by others. Sodium salicylates and antipyrin have been advocated by Pepper. Many different means have been devised of late to cut out the possible auto-toxic agent in the treatment of epilepsy.

The influence which meteorological conditions have upon

epilepsy has been studied by Sokolow for two years. He infers that there are laws in operation between earth magnetism and epilepsy, and between the seasons of the year and frequency of attacks. There are so very many modifying agencies of the every day life of the epileptic which might influence the epilepsy favorably or unfavorably that it is well nigh impossible to fathom the meaning of statistics in regard to the relationship of the meteorological conditions to that of epilepsy. Sokolow found that the seasons favorable for fewness of attacks in the following order: Spring, autumn, summer, winter. This author intends to report more at length at some future time.

Certain authors maintain that infectious diseases have so decidedly a favorable influence over a pre-existing epilepsy that they have advocated the establishment of some of these different processes, such as malaria and erysipelas, to cure the epilepsy. Bourneville, Seglas, Pellissier, Toulouse and Marchand, and Marandon de Montyel have all handled the subject from different view points and arrived at different opinions. In these different studies infectious diseases are proven to be both harmful and beneficial to the epilepsy. The reviewer has known many instances of both results and thinks the effects are so uncertain that it is the part of a wise physician to counsel his epileptic patient to avoid any infection whenever and wherever possible. Hessler, of Indianapolis, is probably entitled to priority in the use of erysipelas serum for the cure of epilepsy. He reports good results from apparently insufficiently elaborated and inextensive experimentation.

In trying to avoid the possible auto-toxics, Maurice de Fleury advocates the use of injections of serum; this increases the action of the bromides to such an extent that he was able to get good results with but three gr. of bromides where formerly one or two drachms were given. The serum is diuretic and relieves the arterial tension, and is antitoxic.

The milk regimen and alterations of the diet have met with signal success in many carefully selected cases of epilepsy. The whole character of life, such as may be embraced in colonization of epileptics, offers to revolutionize the treatment of epileptics. The many forms of treatment, old as well as new,

can only be put in practical operation when the patients are properly colonized and brought under careful observation and regulation.

#### SURGICAL TREATMENT.

In looking over the literature one is much impressed that trephining for the cure of epilepsy is decidedly on the decline. A few cases have been reported of cure by trephining, but the per cent. is very small, not more than 2 or 3 per cent. of those operated upon. The reason for this decline of trephining as a corrective measure is obvious. The *predisposition in epilepsy is paramount*, and trephining does not strike the root of the evil. When the excitant is of the nature of a direct trauma to the cerebral cortex, is recent, and the hereditary factors are not great, trephining may do some good. Very many cases are made worse by trephining, which is a fact that should be carefully borne in mind by the physician before advising operation. Cases rendered worse by operation have been recently reported by Spratling, Bourneville and Rellay.

Removal of the ovaries for cure of epilepsy, and, in fact, all gynecological procedures for treatment of epilepsy, have very little to recommend them; nevertheless Love has recently reported a cure by removal of the ovaries. The reviewer has seen several cases in which the physical and mental condition, as well as the epilepsy, were rendered worse by such an operation. Surgical interference in the epileptic should call for the same treatment as another not afflicted with epilepsy.

As to the surgical procedure of resection or section of the cervical sympathetic, it is destined to fall into deserved disuse. Many experiments and investigations have been undertaken within recent years, and in the vast majority of instances, even when such workers were willing and desirous of finding good results from the practice of the operation, they report unfavorably to its further trial. Many writers maintain, with apparent justice, that the operation is as little indicated theoretically as practically.

#### CONCLUSIONS.

The etiology and pathology of epilepsy seem as far as ever from being solved. The predisposition, hereditary or acquired, has not been carefully investigated of late. The study

of this should be along the lines of physiological chemistry and physiologic psychology; to be directed not so much against the immediate excitants of epilepsy as to the inherent or acquired instability of the cortical centers of epileptics. Thorough and exhaustive studies must be made upon the predisposition before the immense mass of alleged excitants can be properly classified and their particular values as causes be determined.

The manifestations of epilepsy are daily becoming more numerous; some do not deserve to be placed in a separate category, being bits of pieces of aborted symptoms of grand mal, others are only coincidences, while still others are referable to perverted bodily functions, particularly liable to occur in any one subject to degeneracy.

As to what constitutes the essential phenomenon of epilepsy or its pathognomonic sign there still remains much doubt. The more carefully the disease is studied, the more frequently "loss of consciousness" is not found to be constant, although probably a derangement or disorder of consciousness is an ever present symptom. Again, as more care is exercised in observation, the more common some motor symptoms are found to exist. It is now held by the majority of neurologists that the sensory and motor elements of an epileptic crisis cannot be separated, and that varying degrees of exhibition of both are found in epileptic seizures. Studies upon the aura and order of muscular invasion have not helped to solve the epilepsy problem as much as was once prophesied, principally because of the indefiniteness of the mooted points about the motor, sensory and association cortical centers.

The phenomena of psychical equivalents and psychic epilepsy are so differently understood and used by many neurologists that, were they less loosely studied and recorded, they would still be of little aid to us in understanding whether there is really a psychical equivalent, a pure psychic epilepsy, or whether such phenomena are but symptoms that are coincident with the epileptic state.

The possibility for the accurate and careful study of temporary epileptic delirium and the light that it would throw upon the insanities were never greater than to-day. Epilep-

tics are being collected in colonies and special institutions for their care, where unsurpassed opportunities are presented for one to see and know the epilepsies in their complete elaboration.

ANGIOLELLA, G.: "Epilepsy with Lesions of the Frontal Lobes" (*Annali di Neurologia*, 1898, fasc. IV, V, p. 277-286).

ALLEN, C. L.: "Senile Epilepsy with Report of Four Cases" (*Med. News*, 1898, LXII, p. 292).

ADLER: "Post-Convulsive Paralysis after Epileptic Attacks" (*Neurol. Centralbl.*, 1898 (17), 691).

ALZHEIMER: "The Pathological Anatomy of Epilepsy" (*Monatsschrift für Psych. u. Neurol.*).

VON BECHTEREW: "Epileptic and Epileptoid Attacks in the Form of Morbid Fears" (*Neurol. Centralbl.*, Dec. 15, 1898).

BOURNEVILLE: "Remote Results of Surgical Treatment of Epilepsy" (*Revue neurol.*, 1898, p. 591).

VON BECHTEREW: "On the Significance of Cardiac Tonics in the Treatment of Epilepsy" (*Neurol. Centralbl.*, 1898, p. 290).

BESSON: "Case of Epilepsy in a Child Aged Six Years" (*Journal de sci. med. de Lille*, 1898, XXI, No. 1).

BUCCELLIS "Relation of Epilepsy to Alcohol" (Paper read before the Royal Academy at Genoa, March, 1898).

BRATZ: "Changes in the Cornu Ammonis in Epileptics" (*Centbl. f. Nervenheilk. u. Psychiatrie*, 1898, p. 91).

BROWN, P. K.: "Study of 100 Cases of Epilepsy" (*Trans. Med. Soc. California*, 1898, p. 261).

VON BECHTEREW: "Choreatic Epileptic Convulsions" (*Münch. med. Wochensch.*, June 7, 1898).

BOND: "Causation of Epilepsy" (*Journal Am. Med. Ass.*, 1898, XXXI, 263).

BOND: "Ipecacuanha in Epilepsy" (*Lancet*, 1898, II, 751).

BOURNEVILLE and DARDEL: "Epilepsy Following Typhoid Fever" (*Progress méd.*, Mar. 19, 1898).

BISCHOFF: "The Motor Paths in Epileptic Attacks" (*Wien. klin. Wochensch.*, Sept. 28, 1899).

BANDROPADHIA: "Epilepsia Gravior; Its Treatment and Cure" (*Indian Med. Rec.*, July 16, 1898).

BALLET: "Occasional Causes of Epilepsy" (*L'Indépendance méd.*, 1898, p. 121).

BREITUNG: "Epilepsy Developed by a Cold Douche" (*Deutsch. med. Woch.*, 1898, No. 39).

BOMBARDA: "The Nights of Epileptics" (*Revista portuguesa de med. e cirurgia praticas*, t. III, No. 31, p. 193, Feb. 1, 1898).

BONNET: "Treatment of Epilepsies" (*Le Dauphine méd.*, 22 année, No. 6, p. 121, June, 1898).

BAYERTHAL: "Our Knowledge of Pseudo-Meningocele, etc." (*Deutsch med. Woch.*, 1898, 3-4).

BRATZ: "Alcohol and Epilepsy" (*Allg. Zeitsch. f. Psychiatrie*, t. LVI, 3, June, 1899, p. 50).

BOUCARUT: "Meningitis with Choreiform and Epileptiform Symptoms" (*Nouveau Montpellier méd.*, t. VII, No. 31, p. 685, July 31, 1898).

BOMBARDA: "Epileptic Impulses" (*A Med. contemporanea*, 1898, No. 12, p. 89 et 93).

BALLET and FAURE: "Epileptiform Attacks Produced by Experimental Tobacco Intoxication" (*Soc. de Biol.*, Feb. 11, 1899).



BALLET: "Hallucinatory Toxic Delirium with Epileptiform Attacks Apparently Brought about by Sulphate of Cinchonidina" (Revue neurol., July 15, 1899).

BONFIGLI, R.: "The Elimination of Methylene Blue in Epilepsy," etc. (Revista sperm. di freniatria, Vol. XXV, fasc. 2, p. 387-400, July, 1899).

BALLET: "Epileptiform Facial Neuralgia and its Treatment" (Bull. méd., No. 26, p. 299, Mar. 29, 1899).

BOURNEVILLE, TISSIER and RELAY: "Two New Cases of Status Epilepticus" (Progrès méd., No. 11, p. 161, Mar. 18, 1899).

BEREZOWSKI: "The Effect of Cranial Depression in Traumatic and Essential Epilepsy and its Physiological-Anatomical Reasons" (Traux de neurol. chirurg., an. IV, fasc. 2, p. 113-131).

BREGMAN, L. E.: "Ambulatory Automatism" (Neurol Centralbl., Sept., 1899).

BECHET: "Biological Conditions of Epileptic Families" (Arch. de Neurol., Mar., 1899, p. 202-209).

BINGSWANGER: "Die Epilepsie" (Special Path. and Ther., Nothnagel, Band XII, 1899).

BINGSWANGER: "Post-epileptic Ocular Exhaustive Phenomena," *op. cit.*

BINGSWANGER: "Exhaustive Post-epileptic Disturbances of Cutaneous Sensibility," *op. cit.*

BINGSWANGER: "Vasomotor Disturbances Which Accompany Epileptic Attacks," *op. cit.*

BINGSWANGER: "Status Epilepticus," *op. cit.* p. 344.

BABINSKIS "The Hoe Phenomenon" (Gaz. des hôp., Nov. 23, 1899).

BALLARD: "Causes of Death in Epileptics" (Thèse de Paris, 1898).

BYTEREF: "Epilepsy and Epileptoid Manifestations by Fright" (Revista de ciencias med. de Barcelona, Feb. 28, 1898).

BOURNEVILLE: "Recherches cliniques et thérapeutiques sur l'épilepsie, l'hystérie et l'idiotie" (1898 and 1899).

BROWN, S.: "Devious Manifestations of Epilepsy" (Med. Rec., April 29, 1899).

BECK, KARL: "Resection of the Sympathetic Ganglia of the Neck" (Chicago Med. Rec., April, 1899).

BULLARD, W. N.: "Classification of Epileptics" (Boston Med. and Surg. Journal, April 6, 1899).

BERGMANN: "Surgical Treatment of Intra-Cranial Disease" (Third Edition, Berlin, 1899, August Hirschwald).

BRATZ: "The Opium Treatment of Epilepsy According to Flechsig" (Allgem. Zeitschr. f. Psych., Bd. LIV).

CHIPAULT: "New Remarks on the Treatment of Epilepsy by Complete Resection of the Superior Cervical Ganglion of the Sympathetic" (Gaz. des Hôp. 1898, p. 416).

CHAMBRELENT: "Reciprocal Influence of Epilepsy and Pregnancy" (Journal de Méd. de Bordeaux, Nov. 5, 1899).

COMBY: "Case of Partial Epilepsy with Hemiparesis of the Left Side" (Arch. d. Méd. et d. Suf., 1898 (1), 227).

CHRISTIAN, J.: "Severe Cerebral Syphilis," etc. (France méd., 45 année, No. 33, p. 513, Aug. 19, 1898).

CLAUS (ANVERS): "Experiments of Injections of Artificial Serum in the Treatment of Epilepsy" (Bull. de la soc. de méd. mentale de Belgique, June, 1898).

CRICCHIO, G.: "A Case of Syphilitic Epilepsy Cured" (Riforma med., 1898, Vol. II, No. 15, p. 177, April 20).

CESTAN and LE SGURD: (Gaz. des hôp., Nov. 23, 1899). "Babinski's Toe Phenomenon in Epilepsy."

CLARK, L. PIERCE: "Epilepsy, Phocomelus of Humerus as Stigmata of Degeneration" (N. Y. Med. Journal, May 13, 1899).

CLARK, L. PIERCE: "Tetanoid Epilepsy" (Am. Journal of Insanity, April, 1899).

CLARK and SHARP: "An Analysis of Forty Cases of Infantile Cerebral Palsy Associated with Epilepsy" (Pediatrics, Vol. VII, No. 7, 1899).

COLOLIAN, P.: "The Toxicity of the Blood in Epilepsy" (Arch. de Neurol., Mar., 1899).

CROCQ: "A Case of Hysterical Jacksonian Epilepsy" (Journal de Neurol., 1899, No. 17, p. 321-325).

DIDE: "Post-epileptic and Post-eclamptic Mental Confusion" (La Tribune méd., 1898, p. 324).

DIDE: "Typhoid Fever in Etiology of Epilepsy" (Independence méd., March 29, 1899).

DE FLEURY, MAURICE: "Medical Treatment of Epilepsy" (Revue neurol., 1898, p. 420).

DUPRE-LEFEBVRE: "Epilepsy of Gastric Origin" (Le Nord médical).

DONATH: "Value of Resection of Cervical Sympathetic in Epilepsy" (Wien. klin. Woch., 1898, No. 16).

DEUTSCH: "Acetonic Epilepsy" (Wien. med. Woch., 1900, No. 5).

LE DUIGON: "Prognosis of Epilepsy in Childhood" (Thèse de Paris, Felix Ilcan, éditeur 1899).

DEVAY, M.: "Epilepsy, a Case of, with Autopsy" (Lyon méd., Jan. 1, 1899).

EDSALL, E. H.: "Some Cases Bearing on Peripheral Causation of Epilepsy" (Med. News, Nov. 18, 1899).

ELSWORTH, R. C.: "Vaginal Hysterectomy in an Epileptic; Operation Following an Attack of Mania; Cure" (British Med. Journal, May 14, 1898, p. 1260).

EBSTEIN, W.: "The Association of Epilepsy and Diabetes Mellitus" (Deutsche med. Wochens., 1898, No. 1 u. 2).

EWALD, R.: "The Experimental Production of Epilepsy" (May, 1898).

FÉRÉ: "The Hungry Evil in Epileptics" (Revue de méd., July 10, 1899).

FÉRÉ: "Laughing Attacks in an Epileptic" (Soc. de Biol., 1898, 430).

FÉRÉ: "Absence of Association of Reflex Movements of the Eyes in Post-epileptic Stupor" (Soc. de Biol., 1898, p. 123).

FÉRÉ: "Attacks of Deafness in an Epileptic" (Soc. de Biol., V. 1898).

FÉRÉ: "Note on the Crossed Femoral Reflex in Epileptics" (Soc. de Biol., 1898, p. 7).

FÉRÉ: "Epileptic Narcolepsy" (Revue de méd., 1898, XVIII, 430).

FÉRÉ: "Epileptic Fears" (Médecine moderne, 1898, p. 185).

FÉRÉ: "Note on the Spontaneous Epileptic Zone in a Cat" (Soc. de Biologie, 1898).

FÉRÉ: "Note on the Cephalic Pulsations of Epileptics" (Revue neurol., 1898).

FÉRÉ: "Tolerance of Elderly Epileptics to Bromides" (Revue de méd., Jan. 10, 1900).

FÉRÉ: "Post-epileptic Dysæsthesia" (La Normandie méd., Feb. 1, 1900); "Accidents of the Epileptic Paroxysm Connected with Muscular Contraction" (Revue de Chirurgie, Jan. 10, 1900).

FÉRÉ and LAUBRY: "Variation in the Mydriatic Action of Atropine in Epileptics" (Soc. de Biol., 1898, 174).

- FÉRÉ: "Epileptic Priapism" (Med. moderne, No. 10, Feb. 4, 1899).
- FERRAMINI, L.: "Auto-intoxication and Epilepsy" (Annali di neurologia, 1898, fasc. IV, V, p. 329-357).
- FERRARINI: "Auto-toxic Epilepsy of Hepatic Origin" (Revue neurol., 1898, 538).
- FRANCK, F.: "Physiological Signification of the Resection of the Sympathetic in Basedow's Disease, Epilepsy, Idiocy and Glaucoma" (Acad. de méd. May 23, 1899).
- FUTCHER: "Auto-intoxication in Epilepsy" (St. Paul's Med. Journal, Sept., 1899).
- FORNI, L.: "Epileptiform Convulsions from Neuralgia of the Trillings Nerve" (Bolletino del Policlinico gen. di Torino, March and April 16, 1898, p. 70 and 81, ob. II).
- GARNIER and SANTENOISE: "A Case of Paramyoclonus Multiplex Associated with Epilepsy" (Annales Medico-psychologiques, 1898, VIII, 392).
- GELINEAU: "'Intangible' Epilepsy" (Revue de psychiatrie, 1898, p. 370).
- GUILLON: "Two Cases of Retarded Epilepsy" (Gaz. med. de Nantes, 16 annee, No. 33, p. 258, June 25, 1898).
- GILBERT: "Jacksonian Epilepsy Cured by Trephining" (Nouveau Montpellier méd. t. VII, No. 20, p. 405, May 14, 1898).
- GALANTE, PIO: "Post-epileptic Albuminuria" (Journal of Mental Science, April, 1899).
- HOCHHAUS: "Premature Calcification of the Cerebral Vessels as a Cause of Epilepsy" (Neurol. Centralbl., 1898, p. 1026).
- HESSLER: "Epilepsy and Erysipelas" (Journal Am. Med. Ass., 1898, XXX, p. 1157).
- HONIG: "Therapy of Status Epilepticus" (Pester med.-chirur. Presse, 1898, p. 1243).
- HUGHLINGS-JACKSON: "Asphyxia in Slight Epileptic Paroxysms" (Lancet, 1899, p. 79).
- HUGHLINGS-JACKSON: "Uncinate Epilepsy" (*Op. Cit.*)
- HERING: "Irritation of the Cerebral Cortex after Section of the Pyramids or Other Portions of the Central Nervous System, with Especial Regard to Epilepsy" (Wiener klin. Wochenschr., Aug. 17, 1890).
- HOUSE: "Some Factors in the Production of Epilepsy" (Buffalo Med. Journal, XXXVII, 822).
- HAMILTON, A. McL.: "Connection of Nocturnal Epilepsy with Retroejaculation of Semen" (N. Y. Med. Journal, 1898, LXVII, p. 566).
- HIGIER: "Pathogenesis of the Motor Paralytic Equivalents of the Epileptic Attack" (Deutsch Zeitschr. für Nervenheilkunde, March 9, 1899).
- HERTER, C. A.: "Notes on the Toxic Properties of the Blood in Epilepsy" (JOURNAL NERVOUS & MENTAL DISEASE, Feb., 1899).
- HASLE: "Bromide of Camphor in Epilepsy" (Thèse de Paris, 1899).
- HUDSON, W. H.: "Traumatic Epilepsy; Case Trephined; Cured" (Liverpool Medico-Chirurgical Journal, Jan., 1899).
- HODGDON, A. L.: "Preputial Reflex Epileptiform Convulsions with Report of a Case" (Alienist and Neurol, Vol. XVIII).
- HELDENBERG: "Post-epileptic Contractures" (Belgique med. 21, July 28, Aug. 4, 1898).
- JONES and CLINCH: "Epilepsy of Cardiovascular Origin" (Lancet, Sept. 16, 1899).
- JONNESCO, T.: "Resection of the Cervical Sympathetic in the

Treatment of Epilepsy; of Exophthalmic Goiter and of Glaucoma," etc. (*Gaz. des hop.*, April 21, 1898, No. 46, p. 424).

JABOULAY, M.: "Treatment of Epilepsy by Section of the Cervical Sympathetic" (*Lyon méd.* t. LXXXVII, No. 9, Feb. 27, 1898, p. 315).

JACKSON and COLEMAN: "Tasting Movements; Dreamy State" (*Brain*, 1899).

JACKSON and COLEMAN: "Softening of the Gyrus Uncinatus" (*Brain*, part 4, 1898, p. 580).

JOFFROY: "The Convulsive Predisposition" (*Gaz. hebdom. de méd. et chirurgie*, Feb. 11, 1900).

KOVALEVSKY: "Epilepsy from the Clinical and Medico-legal Point of View" (*Annales medico-psychologiques*, 1898, Vol. VII).

KELLNER: "On the Result of the Flechsigs Opium-Bromide Treatment" (*Deutsch med. Wochen.*, 1898, p. 11).

KRAFFT-EBING: "Typical Delirium in Epileptics" (*Arbeiten aus dem Gesamtgebiet der Psychiatrie und Neuropath.*, 1898, III).

KRAFFT-EBING: "Epileptic Psychoses" (*Op. Cit.*).

KRAUSE: "Rare Form of Petit Mal" (*Münch. med. Wochen.*, 1898, p. 1103).

KINGDOM: "Diagnosis of Hystero-epilepsy from Status Epilepticus" (*Lancet*, 1898, II, 320).

KARPLUS: "Differential Diagnosis of Hysteria and Epilepsy" (*Jahrb. f. Psychiatrie*, 1898, Bd. XVI).

KLEIN, E. G.: "Urine in Epilepsy" (*N. Y. Med. Journal*, Dec. 30, '99).

KOCHER, T.: "Epilepsy, a Few Indications for Surgical Treatment" (*Archiv. für klin. Chirurgie*, Band 59, Heft 1).

KOENIG: "Epilepsy and Hysterical Convulsions in Paralysed and Non-Paralysed Idiot Children" (*Monatsschr. für Psych. u. Neurol.*, 1899, Bd. IV).

KOVALEFSKY: "A means of Preventing Epileptic Attacks" (*Messenger med. russe*, 1899, t. I, No. 9, p. 16-17).

KRANSKY: "Pathology of Epilepsy" (*The Alienist and Neurologist*, Oct., 1898, Vol. XIX, p. 511).

LEMOINE: "Gastric Epilepsy" (*Soc. de Biol.*, 1898, p. 65).

LOVE, J. H.: "A Case of Epilepsy Relieved for Two Years by Removal of the Ovaries" (*Southern California Practitioner*, 1898, XIII, 48).

LANNOIS: "Melanoderma in Epileptics" (*Revue neurol.*, 1898, p. 590).

LANNOIS and JABOULAY (de Lyon): "Treatment of Epilepsy by the Excision of the Sympathetic" (*Revue neurol.*, Aug., 1898).

LANNOIS and BERNOUD: "Enormous Angiomatous Nævus of the Face with Spasmodic Hemiplegia and Epilepsy" (*Nouv. iconographie de la Salpêtrière*, t. XI, No. 6, 1898).

LANNOIS and MAYET: "Post-paroxysmal Albuminuria in Convulsive Epilepsy" (*Lyon méd.*, 31 année, t. XCI, No. 29, July 16, 1899).

LANNOIS: "Retropulsive Epilepsie" (*Lyon méd.*, 31 année, t. XCI, No. 30, July 23, 1899).

LEMONS: "Symptomatic Epilepsy from Cortical Lesions" (*Nouv. iconogr. d. l. Salpêtr.*, 1898, p. 20).

LABORDE: "Experimental Epilepsy not Prevented by Resection of the Entire Cervical Sympathetic" (*Gaz. d. hop.*, 1898, 1267).

LUI, AURELIO: "Alkalinity of the Blood in some Psychopathics and in Epilepsy" (*Revista sperimentale die freniatria*, 1898, Vol XXIV, p. 1-19).

LUNDBORG: "On Paramyoclonus Multiplex (family type)" (*Hygica*, Jan., 1899, p. 1).

LESZYNSKY: "Some of the Essential Features in the Diagnosis and Treatment of so-called Idiopathic Epilepsy" (*Med. Record*, May 20, 1899).

LAPINSKY: "Epilepsy in Frogs" (*Pfluger's Arch.* Bd. LXXIV).

LONDE: "General Epileptiform Symptoms in the Course of Hemiplegia" (*Arch. générales de méd.*, July, 1899).

LAPINSKY: "Contribution to the Study of the Excitability of the Cerebral Cortex and Epileptic Convulsions in Lower Animals" (*Moniteur neurol.*, 1899, t. VII, fasc. I, p. 59-77).

LINKE (Zost): "On the Treatment of Epilepsy by Opium and Bromide" (*Allg. Zeitsch. f. Psychiatrie*, t. LV, f. 3, Aug., 1898).

MONTYEL, DE, MARANDON: "Malarial Infection and Epilepsy" (*Revue de Méd.*, Dec., 1899).

MARBURG: "Epilepsy from Abuse of Coffee" (*Wiener klin. Rundsch.*, May 21, 1899, p. 337).

MATTHIOLUS: "Operation on the Skull for Epilepsy" (*Deutsch Zeitschrift für Chirurgie*, June, 1899, Band 52, Heft 1 u. 2).

McKENDRICK, J. S.: "Remarks on Jacksonian Epilepsy, Illustrated by an Unusual Case" (*Brain*, Part III).

MONDIO, GUGLIEMO: "Jacksonian Epilepsy" (*Academie Peloritaine de Messine*, June 4, 1898).

MABILLE: "Religious Hallucinations and Transitory Religious Delirium in Epilepsy" (*Ann. medico-psychol.*, Jan., 1899).

McCONNELL, J. W.: "Transient Paralysis as an Epileptic Equivalent" (*JOURN. NERVOUS & MENTAL DISEASE*, June, 1899).

MURPHY: "Apparent Cure of Idiopathic Epilepsy by the Wearing of Prisms" (*Annals of Ophthalmology*, 1898, VII).

MAIRET AND VIRES: "Toxicity of the Blood-serum of Epileptics" (*Soc. de Biol.*, 1898, p. 678).

MAEWSKI: "Brain of an Epileptic Dying of Status" (*Revue neurol.*, 1898, p. 386).

MAVROJANNIS: "Toxicity of the Sweat in Epileptics" (*Revue de Psychiatrie*, 1898, p. 199).

MARCHAND: "Pulse and Temperature in Epileptic Vertigo" (*Rev. de Psychiatrie*, 1899, p. 244); "Status Epilepticus of Vertigo" (*Rev. de Psychiatrie*, 1899, p. 244).

MAUNIER: "Treatment of Epilepsy by Trional" (*Archives de Neurol.*, March, 1899).

NAWRATSKI and ARNDT: "Variations in Pressure in the Cerebro-spinal Cavity in Convulsions" (*Berl. klin. Wochen.* 1899, No. 30).

OSSIPOW: "Contractions of the Stomach, Intestine and Bladder during the Epileptic Paroxysm" (*Deutsch. Zeitsch. f. Nervenheilkunde*, 1899, Bd. XV, No. 1-2).

OHLMACHER: "Persistent Thymus and Other Morbid Anatomical Peculiarities in Certain Cases of Epilepsy" (*Columbus Med. Jour.*, 1898, Aug.); "Secondary Epilepsy and Multiple Cavernous Angioma," etc. (*JOURN. OF NERVOUS & MENTAL DISEASE*, July, 1899); "Clinico-Pathological Study of Six Cases of Epilepsy" (*Bulletin Ohio Hospital for Epileptics*, Jan., 1898), (*Bulletin of Ohio State Hospital for Epileptics*, Vol. I, 1899, Nos. 2, 3 and 4).

POPOFF: "A Case of Hereditary Epilepsy" (*Messenger méd. Russe*, 1899, t. I, No. 17, p. 1-16 et No. 18, p. 1-18).

PELISSIER, L.: "The Influence of Intercurrent Infectious Diseases on the Progress of Epilepsy" (*Thèse de Montpellier*, 1898).

PREOBRJINSKY and SARYTCHEFF: "A Case of Moral Insanity and Epilepsy" (*Memoires méd. Russe*, 1899, VI).

PEPPER: "A Case of Epilepsy Treated for Eight Years with Antipyrine and Sodium Salicylate" (*Southern Practitioner*, 1898, XX, 58).

PRUS: "Pathogenesis of Epilepsy" (Wien. klin. Woch., Sept., '98); "Investigations upon the Electrical Excitability of the Corpora Quadrigemina" (Wiener klin. Wochens., 1899, No. 45); "The Origin and Course of Cortical Epilepsy" (Wien. klin. Wochens., 1898).

PATRICK, H. T.: "Epileptic Child and His Future from a Medical Standpoint" (Feb. 11, 1899). (Phila. Med. Journ.)

PUTNAM and PFAFF: (Boston Med. & Surg. J'l. April, 1899).

PICK: "Relations between Epilepsy and Sleep" (Wien. med. Wochens., July 22, 1899).

REGIS, PEON: "Report of Epilepsy and General Paralysis" (Revue neurol., 1898).

ROCHE, ANTHONY: "Strontium Bromide in Epilepsy" (Lancet, April 22, 1899).

RICHTER, R.: "Sensorial Disturbances in Hysteria and Epilepsy" (Archiv. für Psych., Band 31, Heft 3).

ROSSI, CESARE: "Excitability of the Cord in the Report with New Therapeutics on Epilepsy" (Rivista sperimentale di freniatria, Vol. XXIV, fasc. II, July 15, 1898).

RAW, NATHAN: (British Med. J'l, May 6, 1899).

RISPAL and ANGLADE: "Condition of the Nerve Cells in a Patient Dead of Status Epilepticus" (Revue neurol., 1898, p. 586).

RACHFORD: "Relationship of Migraine to Epilepsy" (Am. Journ. Med. Sci., CXV, p. 436).

ROWLAND, WALTER J.: "Case of Plumbic Epilepsy; Prolonged Status Epilepticus; Recovery" (British Med. Journ., April 16, 1898, p. 1013).

RENNIE: "Muscular Hypotonia in Epileptics" (Lancet, July 15, '99).

RELLAY: "Craniotomy, etc., for Epilepsy" (Thèse de Paris, 1898), (Felix Alcan éditeur).

ROHRMANN: "Treatment of Epilepsy with 'Bromalin'" (Monatsschr. f. Psych. u. Neurol., Bd. IV).

REYNIER: "Trephining for Jacksonian Epilepsy" (Soc. de Chirur., March, 1899).

RABE and MARTIN: "Epithelioma of the Breast. Mediastinal and Intra-Cranial Cancer by Metastasis. Symptomatic Jacksonian Epilepsy" (Soc. anatomique de Paris, Dec. 25, 1898).

STERN, H.: "Alcoholic Epilepsy" (Quar. Journ. Inebriety, 1898, p. 81.)

SINKLER, W.: "Long Remissions in Epilepsy and their Bearing on Prognosis" (JOURN. NERVOUS & MENTAL DISEASE, Aug., 1898).

SCHROEDER: "Treatment of Flechsig" (Centralbl. f. Nervenheilkunde, Aug., 1898, p. 451).

SPRATLING, W. P.: "Epilepsy, a Remarkable Case" (N. Y. Med. Journ., March 18, 1899); "Curability of Epilepsy" (Medicine, Feb., 1900); "Trephining in Epilepsy" (Phila. Med. Journ., Jan. 28, 1899).

SUCKLING: "Treatment of Epilepsy" (Birmingham Med. Review, March, 1899).

SCHAPIRO: "Study of Epilepsy; Its Treatment by Resection of Great Sympathetic" (Thèse de Paris, 1898).

SILVA, B.: "A Case of Jacksonian Epilepsy; Contribution to the Study of the Localization of the Center of the Superior Facial" (Il Policlinico, Vol. V-M, f. 9, p. 393, Sept. 1, 1898).

SPILLMANN, LOUIS: "Lardaceous Scleroderma as a 'Sabre Cut' in the Frontal Region. Concomitant Epileptiform Attacks" (Nouv. icon. de la Salpêtrière, May, June, 1898, No. 3).

SCOBBO: "The Electric Current in the Treatment of Epilepsy" (Giornale internaz. delle scienze med., an. XX, fasc. 2, 1898).

STANLEY: "Case of Post-epileptic Automatism" (*Intercolon. Med. Jour.*, 1898, III, 104).

SOKOLOV: "Influence of Meteorological Conditions (Especially Magnetism of the Earth) upon Epileptic Paroxysms" (*St. Petersburg med. Wochens.*, 1898, No. 15).

SCHUSTER and MENDEL: "Epilepsia Cursoria" (*Münch. med. Wochens.*, July 11, 1899).

SMITH, J. G.: "Bromide of Strontium in Epileptic Insanity" (*Lancet*, Aug. 12, 1899).

SALGO: "State of Consciousness in Epileptic Attacks" (*Allg. Zeits. f. Psych.*, t. LXI, f. 1, 2, May, 1899).

SPINHAYER: "The Treatment of Epilepsy by the Combination of Adonis Vernalis and Bromides" (*Bull. de la Soc. de méd. mentale de Belgique*, Dec., 1898).

SOUQUES, A.: "Unsuccessful Treatment of a Case of Epilepsy by Bilateral Resection of the Cervical Sympathetic" (*Revue neurol.*, July 15, 1899).

TEDESCHI: "The Cerebral Gliosis of Epileptics" (*Il Policlinico*, an. VI, Vol. VI, 1899).

TOULOUSE and MARCHAND: "Delirium as Equivalent of Convulsive Attacks in an Epileptic" (*Soc. de Biol.*, Jan. 7, 1899).

TODD: "Note on the Management of the Toxic Element in the Treatment of Epilepsy" (*Chicago Med. Recorder*, 1898, XIV, 337).

TURNER, ALDREN: "Colonies of Epileptics" (*Soc. Harveienne de Londres*, April 14, 1898).

TOULOUSE and MARCHAND: "Influence of Infectious Diseases upon Paroxysms of Epilepsy" (*Revue de Psych.*, 1899, p. 123); "General Paralysis of Youth and Epilepsy" (*Revue de Psych.*, July, 1899).

VIGNAUD: "Case of Epilepsy" (*Lyon méd.*, 1898, Vol. 87, p. 51).

VOISIN: "Elimination of Methylene Blue in Epileptics" (*Revue neurol.*, 1898, 590).

VIGNARD, P.: "Fall on the Head. Epilepsy Dating from Six Years of Age. Trephining. Cure" (*Lyon méd.*, t. LXXXVII, No. 2, p. 51, Jan., 1898).

VIDAL, E.: "Influence of the State of Cerebral Circulation in the Production of Experimental Toxic Epilepsies" (*Soc. de Biol.*, March 18, 1899).

VARIAT, G.: "A Case of Congenital Cyanosis with Epileptiform Paroxysmal Attacks" (*Journ. de clin. et de therap. infant*, No. 5, Feb., 1898).

WRIGHT: "Effects of Poisonous Doses of Potassium Bromide on the Cerebral Cortical Cell" (*Brain*, 1898).

WEBER, L. W.: "Results of Autopsy in Status Epilepticus" (*Wiener med. Wochens.*, 1899, No. 4).

WEBER: "The Role of Auto-intoxication in Epilepsy" (*Münch. med. Wochens.*, 1898, p. 813).

WISLOCKI: "Study of Epilepsy and Its Treatment" (*Kronika lekar-ska*, March 15, 1899; *Arch. de Neurol.*, 1899, p. 133).

WITTNER: "Chronic Diffuse Post-epileptic Encephalitis" (*Allg. Wien. med. Zeitung*, Sept. 6, 1898).

WACHENHEIM: "Atropine in Epilepsy" (*N. Y. Med. Journ.*, July, 1899).

WARDA: "Treatment of Epilepsy by Opium Bromide" (*Monatsschr. f. Psych. u. Neurol.*, Bd. 11, s. 257).

WILDERMUTH: "On the Care of Epileptics" (*Irrenpflege*, 1898, No. II u. 12).

ZIEHEN: "Questions of the Day Regarding Epilepsy" (*Revue neurol.*, 1898, p. 813).

Author unknown: "Epilepsy from Poisoning with Carbon Monoxide" (Allg. Wien. med. Zeitung, Oct. 10 and 17, '99).

Editorial in Journ. (Am. Med. Ass., Vol. XXXI, p. 611): "Curability of Epilepsy."

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- 76 UEBER DEN SCAPULO-HUMERALREFLEX (The Scapulo-Humeral Reflex). W. v. Bechterew (Neurol. Centralb., March 1, 1900, No. 5, p. 208).

As scapulo-humeral reflex v. Bechterew designates one which he has been able to obtain with fairly marked constancy by tapping with the percussion-hammer upon the inner border of the scapula in the neighborhood of its inferior angle. It consists generally in adduction of the corresponding humerus toward the trunk, often also in an outward rotation of slighter degree, chiefly produced by contraction of the infraspinatus and probably the teres minor. Not infrequently, however, this same reflex, since it may extend to the territory of the deltoid and the biceps, leads to abduction of the arm and moderate flexion of the forearm. It is probably represented in the cord in the neighborhood of the cervical enlargement.

V. Bechterew's belief in the constancy of this reflex has already been mentioned, so that he regards its bilateral disappearance of great importance in certain cases where the other reflexes of the upper extremities and of the trunk are present or abnormally lively. For the same reason its unilateral diminution or complete absence is of equal significance.

In cases of poliomyelitis and in the spinal type of progressive muscular atrophy, if the shoulder-girdle musculature be affected, v. B. has always found this reflex absent, and the same obtains in neuritides involving the shoulder region. In the muscular dystrophies he has found the reflex to diminish progressively with the increase in the dystrophy; and in the condition described by him as "steifigkeit der Wirbelsäule" it is generally decreased. On the other hand, in cases of cerebral hemiparesis and hemiplegia the reflex appears, as a rule, to be markedly increased, even where these affections are accompanied by pronounced atrophy of the shoulder-girdle muscles. From this last characteristic the author believes the reflex to be of importance in determining (in obscure cases) whether an atrophy of the above sort be of cerebral, spinal or neuritic origin.

J. W. COURTNEY.



## Society Reports.

### NEW YORK NEUROLOGICAL SOCIETY.

April 3, 1900.

The President, Dr. Frederick Peterson, in the Chair.

#### THYROIDECTOMY FOR GRAVES' DISEASE.

Dr. B. Farquhar Curtis reported this case, and presented the patient. The patient had been operated upon in November, 1897. She had had symptoms for some time previously, and the goiter was of moderate size. The eyes were quite prominent, and the pulse ranged from 120 to 150. Primary union had followed the operation. There had been no interference with the general health, and improvement had been steady since that time. The left half of the thyroid had been removed. Less than two months after the operation the woman had been able to resume her occupation of bookkeeper, and her pulse had gradually become slower. Apparently the cure was a permanent one.

#### RESECTION OF THE SYMPATHETIC FOR GRAVES' DISEASE.

Dr. Curtis also reported this case, and presented the patient, a woman of twenty-nine years. She had had three pregnancies, and the disease had been worse during each pregnancy. The symptoms had lasted about four years before operation, and had been quite marked during the last two years. In December, 1898, she had a moderate exophthalmos, a rather rapid pulse, and some nervousness. In May she developed a paralysis of the left vocal cord, and twelve days later Dr. Curtis operated upon her at Bellevue Hospital, removing the cervical sympathetic on both sides, but leaving the goiter untouched. While she was in the hospital the pulse had at no time been above 106. Since the operation the nervousness had diminished, and the eyes had improved decidedly. The first effect of the operation had been a reduction in the size of the gland.

Dr. Theodore Janeway said that when she had returned to him last January the thyroid had increased in size, the pulse had been about 90, and aortic and systolic murmurs had developed, and she suffered a good deal from headache. Examination of the urine was negative.

Dr. Curtis said that he had done nine thyroidectomies for this disease, and in none had the operation itself been complicated in any way, not even by hemorrhage. Of the nine cases, six had recovered, and three had died. The first of the fatal cases was that of a woman who had been in fair condition at the time of operation, but had become stupid immediately thereafter, and had developed a temperature of 107° F. before death. Death had seemed to be chargeable to thyroid poisoning rather than to uremia. In the second fatal case the symptoms had been quite severe, and the exophthalmos marked. The patient had not suffered much from shock, but the day following operation

she became very restless, and the temperature and pulse rose rapidly. She suddenly succumbed, apparently from heart failure. The third fatal case was that of a woman of twenty-three years, apparently in excellent condition, with a moderately large goiter and much nervousness. She stood the operation well, but thirty hours later developed a very rapid pulse and respiration, together with a high temperature and great nervous excitement. A little albumin was found in the urine. In all three cases there had been nothing, from the surgical standpoint, to criticize; hence, he believed the fatal result was attributable to thyroid poisoning. He had never seen such symptoms in an ordinary case of thyroidectomy. It was worthy of note in one of the cases, marked by a pulse varying from 120 to 150, the pulse had dropped to 100 before the patient had left the operating table, and had since then varied between 90 and 100.

Dr. Graeme M. Hammond said that the results obtained by Dr. Curtis seemed to point very strongly to the majority of these cases being the result of a hyperactivity of the thyroid gland. It was not improbable that some special nervous condition was the underlying cause of this hyperactivity of this gland. He thought one should hesitate before advising thyroidectomy in cases of exophthalmic goiter in view of a mortality of 33.1-3 per cent. It would be interesting to know the effect of the administration of thyroid extract in cases exhibiting these toxic symptoms after operation.

Dr. E. D. Fisher said that he had seen a patient in whom the cervical ganglia had been resected, and her general condition had certainly improved. A number of these operations had been reported, and it seemed to be a justifiable procedure. Some operators had reported the occurrence of considerable hemorrhage at the operation, and had explained it by the marked dilatation of the blood vessels, supposed to be produced through the cervical sympathetic. In some of the cases relief had followed even removal of one ganglion. On the removal of each ganglion in the case just presented a very transient dilatation of the pupil on that side had been observed.

Dr. W. M. Leszynsky said that the prognosis of Basedow's disease had always seemed to him to depend largely upon the financial ability of the patient. It was for this reason that patients of the poorer class found it desirable to submit to operation. He had tried repeatedly, and in vain, to secure the admission of such patients to our city hospitals for a prolonged course of treatment.

Dr. Fisher thought many of these cases did not respond to even the best methods of treatment, and under the most favorable conditions.

Dr. Leszynsky contended that the early resort to proper treatment, an essential feature of which was prolonged rest, would cure very many cases.

Dr. Peterson took the same view as the last speaker. Both operations described by Dr. Curtis seemed to him very serious ones to undertake until safer methods of treatment had been given a fair trial.

Dr. Curtis said that in his cases the opinion had been expressed at the time, that the poisonous symptoms had been the result of the escape of too much thyroid secretion into the system. It was possible that earlier operation would eliminate this danger.

#### WORD-DEAFNESS WITH SPECIAL REFERENCE TO THE NAMING CENTER.

Dr. Graeme M. Hammond, who read this paper, said that Broadbent had first brought forward, as a theoretical propo-

sition, this "naming center," but it had not been placed on a more tangible basis until a case had been carefully observed and reported by Mills. Dr. Hammond said he had suggested that the cells which were the most highly organized would be the ones which would naturally retain and register the names. If this were the case, the location of the centers in which the names were stored might differ materially in different individuals. Further study, however, had led him to think that such a theory was not necessary to a proper understanding of the cases already reported by others, as well as by himself. Dr. Hammond then presented an illustrative case—that of a man who had been injured in a fight on May 8, 1899. He had been struck on the temple, and had been able to walk home, but a few hours later had had a severe general convulsion, and had lost the power to name objects. He could talk voluntarily, and could say most words except the names of objects and persons. He recognized familiar objects, as evidenced by gestures, but he could not give their names or repeat the names after they had been told him. Dr. Seneca D. Powell had operated upon the man, and had found a linear fracture of the temporal bone, and a clot which completely covered the superior temporal convolution. At about the junction of the posterior third with the middle third was a hole about the diameter of an ordinary lead pencil, and fully one inch in depth. The man made an excellent recovery from the operation, and soon regained the power of naming objects, but the word-deafness had never entirely disappeared. At the present time he found it difficult to pronounce words of three syllables or more.

Dr. Hammond also reported a case in which there had been softening of the middle third of the second temporal convolution. The patient had entered the Charity Hospital on January 9, 1900. He was about forty years of age, and had a fair education. He was comatose at the time of his admission, but soon recovered in large part his normal mental condition. There was no motor weakness, and disorders of sensibility were not noted. The special senses appeared to be unimpaired. He was almost completely word-deaf, was completely word-blind, and was absolutely agraphic. He could not write from dictation nor copy simple geometric figures. He was, however, by no means stupid. Had he lived, a diagnosis of a lesion of the superior temporal convolution, and of the angular gyrus would have been made, and yet neither of these regions had been implicated at all, as shown by the autopsy. In the first case, loss of power to name objects did not depend primarily upon the lesion. In the second case, the region

implicated was almost outside of the accepted region for the naming center. In Mills' case, word-blindness had been associated with anomia, and yet the lesion had been further from the higher visual center than in the second case just reported. The speaker thought the presence of word-blindness or word-deafness, either alone or in combination, only implies that a lesion in any part of the speech area may give rise to various forms of aphasia. It seemed probable that any lesion disturbing any part of the speech area might cause anomia, and hence it could be understood how two such widely separated lesions as in Mills' case and in his own could produce like results.

Dr. E. D. Fisher suggested as a possible explanation that in the case just reported the lesion had been more deeply seated than in Mills' case—not in the cortex proper, but in the fibers leading from it.

Dr. Joseph Fraenkel said that the patient presented seemed to understand the meaning of the words pronounced, the defect seeming to be rather motor than a pure word deafness. It, therefore, seemed to him that the man had recovered from his aphasic disorder. Concerning the existence of a naming center, Dr. Fraenkel said that he agreed with Dr. Hammond that there was no distinct naming center. He had at present under observation a syphilitic man, about forty years of age, who had been recently exhibiting impaired mental faculties and a spastic paralysis of the right upper extremity. In addition, he was absolutely unable to name an object shown him, but he could define the uses of the object. In this case he felt convinced that there was one sufficiently large lesion in the arm center to encroach slightly upon the speech center. A mild interference with the speech area would lead to marked disturbance of the higher and more complex functions associated with speech.

Dr. Hammond said that in the case shown this evening the probability of any injury to the connecting paths between the centers was rather remote. If there were any such interruption it should have been the higher auditory and higher visual centers, which was not the case. In Mills' case the lesions were in the third and fourth convolutions, and he knew of no conducting fibers here which would have been affected. He wished to emphasize the fact, that in a given case of word blindness or word deafness one could not say definitely what is the location of the lesion further than that it involved some part of the speech area.

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77 SYNDROME D'ERB (Erb's Syndrome). D. de Buck (*Journal de Neurologie*, No. 4, 1900, p. 61).

Sixty-nine cases of Strümpell's asthenic bulbar paralysis have been collected from the literature by de Buck. To these de Buck adds a case of his own and three cases mentioned in a supplement to his paper. In his case the symptoms were general paresis, a feeling of general fatigue increased by movement or mental exertion, intermittent course, with variations from day to day, and atrophy with diminution of the faradic contractibility in the muscles of the scapular region and arms. Distinct disturbance of sensation, fibrillary tremor and qualitative changes in the electrical reactions were not noticed. Muscular atrophy has been described in several cases of the syndrome of Erb.

SPILLER.

## CHICAGO NEUROLOGICAL SOCIETY.

January 18, 1900.

The President, Dr. Richard Dewey, in the Chair.

### HEREDITARY CEREBELLAR ATAXIA.

A young man with this affection was shown by Dr. Hugh T. Patrick. The patient was almost eighteen years of age and had developed in a perfectly normal way until the age of fourteen. He had attended the public schools where his progress had been satisfactory, and he had then taken a position as messenger and general utility boy in a store where his services were also satisfactory. The first symptom to appear was clumsiness of the legs, and this was soon followed by a gradual change in disposition and some impairment of memory. From this time there had been gradual mental and physical failure until mentally the condition was almost one of imbecility, and physically the patient was quite incapacitated for any employment. He walked with a wide, uncertain gait, the pupils were slightly irregular and did not react to light and not perfectly to accommodation. Speech was rather slow, somewhat indistinct and inco-ordinate, and accompanied by over-action of the facial muscles. The deep reflexes were all exaggerated with the exception of the jaw jerk, which could not be obtained.

An elder brother had died at the age of twenty-one of a similar disease, which began when he was about fifteen years old.

### AN ETIOLOGIC STUDY OF NEUROSES OF PERIPHERAL ORIGIN.

Dr. H. Gradle read a paper with this title, objecting to the name "reflex neuroses" as inappropriate; the speaker classified under the head of neuroses of peripheral origin those nervous disturbances, sensory or motor, which are maintained by some peripheral anomaly, and which cease when the peripheral cause is removed. As examples, he quoted headache and migraine due to ocular defects or to nasal disease, the neuralgias due to disease of the accessory sinuses or disease of the teeth, the asthma of nasal origin, the laryngeal spasm of pharyngeal origin. Epileptoid convulsions are sometimes, but very rarely, of reflex origin. The symptoms of exophthalmic goiter have been observed a few times as a nasal neurosis. Choreic spasms of the eyelids are probably the result often of chronic conjunctivitis, although this is not positive. According to the speaker's experience they are not due to refractive errors. Neurasthenia is not a neurosis of peripheral origin in any case; on the contrary, it is the underlying basis of many instances of such neuroses. When a peripheral source of annoyance is re-

moved the neurasthenic manifestations are of course benefited.

The fact that errors of refraction usually cause in healthy people merely fatigue and blurring of the eyes, but in certain patients bring on disturbing nervous symptoms, can only mean that in the latter instance other factors are involved as well as the eye-strain. These influences predisposing to neuroses of peripheral origin can often be ascertained from the patient's history, but not always. They were enumerated and described in detail under the heads of "heredity," "insufficient nutrition during the nursing period," "want of outdoor exercise and inactivity of muscles," "loss of sleep," "mental worry;" in short, unhygienic surroundings. Anemia is a very frequent determining condition. Disturbances of the intestines sometimes seem to predispose to ocular neuroses, and occasionally such neuroses persist on a psychic basis after the intestinal disturbance has disappeared. The gist of the paper was to the effect that the concurrence of various factors was necessary in the production of neuroses of peripheral origin, as well as the existence of a peripheral anomaly, and that these, hence, should also receive due attention.

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- 78 INSUFFISANCE NASALE HYSTERIQUE (Hysterical Nasal Insufficiency). Marcel Lermoyez (La Presse médicale, Jan. 25, 1899, p. 37).

The patient, a young girl, had had in childhood complete obstruction of the nasal fossa, as a result of hypertrophy of the inferior turbinated bones. This was relieved, and although the nasal cavities were completely clear, she was utterly unable to respire through them, and if the mouth were held forcibly closed, rapidly manifested signs of asphyxia. There was no spasm of the palatal muscles which closed the anterior fossa of the nose; in fact, the air passages were completely free. Although the patient had never had any crises or other of the ordinary manifestations of hysteria, she was carefully examined for stigmata, with the result of finding complete anesthesia to all forms of sensation over the whole body, and extreme concentric refraction of the field of vision, with preservation of the normal pupillary reactions; complete loss of taste and smell, and loss of ability to perceive sound by bone conduction. There were no hysterogenic areas, and no emotional manifestations. The patient was, in fact, of a gay and active disposition. It was now found that the mere pretense of closing the mouth when the lips were held sufficiently separated to allow the passage of air produced a cyanosis as profound as that produced by complete closure of the mouth. The patient was observed during sleep, and it was found that the nasal breathing was perfectly normal, the mouth remaining closed. Lermoyez explains the case as probably the result of an auto-suggestion at the time of operation, that nasal breathing would be impossible, it being his belief that the effort to breath by the nose required a synergy of muscular effort that could only be carried out by sustained attention and will-power, and that of both of these the hysterical patient was incapable. SAILER.

## Periscope.

### CLINICAL NEUROLOGY.

- 79 "CASUISTISCHE MITTHEILUNGEN ÜBER SCHÄDEL- UND GEHIRNVERLETZUNGEN" (Cases of Severe Skull and Brain Injury). Frölich, (Münchener medicinischer Wochenschrift, 1900, No. 6).

The author reports two cases, the first of which is interesting as showing how severe an injury the brain may suffer without a fatal result.

A boy of 14 fell into a shaft 30 m. deep, the cover of the shaft falling after him, and striking him on the head. He was picked up and brought to the hospital, completely unconscious, with left hemiplegia. On the right side of his skull there was an opening 6 cm. long and 2 cm. broad, extending from the lower half of the anterior border of the parietal bone to the neighborhood of the junction point of the parietal, frontal, and temporal bones, somewhat obliquely downward and forward. From this opening protruded a mass of brain matter and blood coagulation, and an even larger portion of brain substance was matted in the patient's hair, ostensibly by reflex movements of the unparalyzed right arm. After cleaning up the neighborhood of the wound and removing the destroyed brain substance, a probe introduced struck against the missing piece of bone in the brain within the skull, where it had been driven edgewise, cutting its way like a knife. Its careful extraction was followed by such a gush of blood that the patient seemed about to die upon the table. By packing with gauze, however, the hemorrhage was controlled, a light dressing was applied and on top of this the ice bag. On the second day the patient began to regain consciousness, and on the third his sensorium was comparatively clear, and he was able to speak and to give some account of the accident. There was complete left hemiplegia. No febrile reaction occurred. On the fourth day the dressing was changed and the tampon removed. There was no more hemorrhage, and the brain was found level with the opening. The patient did not seem to suffer any pain.

Later the brain showed a tendency to bulge out of the opening, and could not be restrained by pressure on account of the severe pain which this caused. Twenty-five days after the accident the bulging having apparently reached its height, the protruding portion of the brain was removed by continuous ligature. The paralysis began to improve, and in three months the patient could get around with the aid of a stick. His mental condition did not seem at all impaired. Four months later he was discharged from the hospital. A year after the accident he had recovered, except for a paralysis of the arm.

The second case is that of a man of 21, who in December, 1888, shot himself in the right temple. The ball was searched for, but not found. After three weeks the patient was discharged from the hospital, the wound healed, but he was blind in the right eye. On November 23, 1890, he was seized with pain in the head and neck. There came on delirium and convulsions, and there was paralysis of the left arm.

His condition grew worse, and he died December 12. The autopsy showed that the ball had passed in along the junction of the great wing of the sphenoid and the frontal bone, and had been deflected into the orbital cavity, where it was found in a mass of callus. The right optic

nerve had been destroyed. All over the base of the brain there was a fibropurulent deposit. It seems remarkable that an inflammatory complication should ensue so long after the original injury. ALLEN.

- 80 UN CAS D'ANOREXIE HYSTÉRIQUE (A Case of Hysterical Anorexia). Georges Gasne (Nouvelle Iconographie de la Salpêtrière, January and February, 1900, 13th Year, No. I, p. 51).

This is a very interesting case of hysterical anorexia in which a remarkable degree of emaciation was produced. A photograph of the case shows it very well. The case is briefly as follows: Young girl, sixteen years old, mother hysterical, father alcoholic. First hysterical attack followed a psychical trauma due to dangerous illness of mother. This attack was accompanied with convulsive seizures and temporary paralyses, followed by amaurosis, hallucinations, and, most marked of all, an exaggerated condition of anorexia. To be noted is the fact that the mammary gland did not take part in the general emaciation. The patient's weight on her entrance to the Salpêtrière was 55 livres (pounds). Under treatment, mainly by isolation, her appetite returned and her weight increased to 80 pounds. The author adds a number of interesting comments upon the case. True anorexia must be distinguished from an anorexia due to pain following the ingestion of food and from the anorexia following vomiting, as the result of this ingestion. The pain and vomiting can be thought of as resulting from the hyperalgesia of the mucous membrane of the stomach. The possibility of discovering if this pure anorexia is due to an anesthesia of the mucous membrane could not be determined in this case, as no sensory disturbance over the abdomen in the gastric region could be demonstrated. It is a well known fact that anesthesia or hyperesthesia of this region is often accompanied by profound digestive disturbances. It is further noted that while the hysterical nature of these anorexias is everywhere admitted, for the most part no definite hysterical stigmata can be demonstrated. Whether such hysterical individuals are monosymptomatic or not, the condition is cured in much the same way as other hysterical symptoms more commonly met with, namely, by isolation. SCHWAB.

- 81 UEBER OBJECTIVE SYMPTOME LOCALER HYPERÄSTHESIE UND ANÄSTHESIE DES SOG. TRAUMATISCHEN NEUROSEN UND BEI HYSTERIE. (Concerning the Objective Symptoms of Local Hyperesthesia and Anesthesia in the so-called Traumatic Neuroses and in Hysteria). W. v. Bechterew (Neurol. Centralbl., March 1, 1900, No. 5, p. 205).

V. Bechterew points out the fact that although local hyperesthesiae are by no means exclusively peculiar to the so-called traumatic neuroses, they nevertheless constitute—together with tenderness on pressure—one of their most constant signs. It is necessary, therefore, particularly in cases where there is a suspicion of simulation, that we should have at command definite and satisfactory methods of ascertaining the presence or absence of these phenomena in a given case, irrespective of the patient's statements; in other words, methods which will yield irrefragable objective proofs. Among these methods v. Bechterew gives a high place to that of Mannkopf, and this in spite of all recent views to the contrary. In using this test v. B. does not limit himself to the observation of the increase in the pulse-rate thus produced, but also studies the variation in the curve of the pulse, simultaneously brought about.

The dilatation of the pupils which is caused by pressure over painful areas he regards as an objective sign of even greater importance than the heart symptoms, since it can be elicited at will and without the aid



of instruments. It is moreover capable of measurement by Weber's compasses and may even be photographed if desirable in a given case.

Another objective symptom of pain on pressure is the marked variation in vasomotor reaction which often follows, and is manifest, particularly when the painful area is in the head, by flushing of the face. The very evident increase in the respiratory movements which sometimes follows manipulation of the painful part is also a sign of no small importance.

In the cases where hemianesthesia is present, the skin reflexes are often diminished on this side, and one is enabled to make important comparisons between the reaction of the pulse, pupils and respiration to painful stimulation on the anesthetic and the sound sides. Hemianesthesia in traumatic as well as in ordinary hysterical cases is accompanied, as a rule, by spasm of the peripheral vessels, whereby differences in body temperature on the two sides, as well as differences in the cutaneous vasomotor reactions, are often to be made out.

If all these methods of examination are carefully pursued, v. Bechterew believes that the necessity of relying upon a patient's statements with regard to pain may be largely eliminated in that important class of cases which involve litigation.

J. W. COURTNEY.

82 UNUSUAL GUNSHOT WOUND OF BRAIN. Barker (*Lancet*, Dec. 2, 1899).

The author reports a case in which a man, with suicidal intent, discharged two shots from a revolver through his mouth upward and backward. For forty-eight days there was occasional vomiting. There was at first no paralysis, but later the power in the left arm and leg was considerably reduced. The temperature was at no time over 100° F. Consciousness was not at any time suspended. After six weeks the vomiting ceased, the slight headache of which he had complained disappeared and the power of the left arm and leg increased so that he was soon able to walk. Both bullets were shown by skiagraphs to lie within the skull. Making due allowance for the different size of the shadows and that of the man's head, the operator's calculations led him to the conclusion that one of the bullets struck against the vertex and fell backward into the median fissure. In describing the effect of the shot, the patient said that he felt the bullet rattle against the vertex. As long as the presence of the bullet caused no serious symptoms Barker concluded that it was best to leave it alone, but on the sixty-sixth day the patient had a convulsive seizure lasting from three to five minutes. Two days later he had a similar attack, and an hour after the second a third; on the following day he had a fourth. On the following day an osteoplastic flap was sawed out and turned back with the base toward the middle line, thus exposing the longitudinal sinus without injuring it. A probe passed along the fissure in the direction in which the bullet was supposed to lie touched the bullet, and it was removed with forceps under the guidance of the little finger. The finger acted better than the probe, as during a coughing spell the latter produced slight injuries to several vessels and to the brain substance. There was, however, very little bleeding. The dura was stitched in place as far as possible and the osteoplastic flap replaced. Consciousness returned rapidly, but sensation and motion were absent from the whole left side, with the exception of the face. They returned slowly, and a month after operation, while there was fairly good sensation, the use of the left arm and leg was still very imperfect. Afterward the power of the muscles greatly improved, but the epileptic fits recurred so that a year after the injury, Chipault, of Paris,

trephined and separated some adhesions between the two hemispheres. For nine days prior to this operation there were from sixty to one hundred epileptic seizures in a day, with complete hemiplegia. After the operation the epileptic attacks disappeared and there was a steady gain in power in the left leg and arm. JELLIFFE.

#### PATHOLOGY.

- 83 HEMIANOPSIE AUF EINEM AUGE MIT GERUCHSHALLUCINATIONEN (Hemianopsia of One Eye with Hallucinations of Smell). Max Linde (Monatsschrift für Psychiatrie und Neurologie, 7, 1900, p. 44).

The symptoms observed in this case were failure of memory, complete loss of sense of smell, right abducens paresis, loss of the outer half of the right visual field for white and colors, with preservation of the fixation point (the visual field of the left eye was normal), presence of Wernicke's hemianopsic pupillary reaction sign—reaction of the pupil of the right eye only when light was thrown on the right half of the retina, the pupillary reaction in the left eye being normal—deviation of the tongue to the right in extension, later bilateral choked disk, hallucinations of smell, etc. The left hippocampal gyrus and uncus were enlarged and hard on account of a tumor within them, and the right abducens was much atrophied, and the left optic tract was three times the normal size from tumor masses contained within it. The inner part of the right optic nerve was stained black by the Marchi method. In the chiasm the crossed right optic bundle, both dorsal and ventral portions according to Henschen's diagram, was degenerated. The degeneration was due to the tumor masses within the left optic tract. This is supposed to be the first recorded case of unilateral hemianopsia from cerebral tumor with necropsy. SPILLER.

- 84 EXPERIMENTELLE BEITRÄGE ZUR PATHOLOGIE DES RÜCKENMARKS (Pathology of Spinal Cord after Embolus). Hoche (Arch. f. Psych. 32, p. 251-975).

In order to get a clearer idea of the results of inflammations and softenings of the spinal cord, the author conducted experiments on animals with that end in view. He describes his failure with injections made in the carotids, as the animals died very soon after of general arterial embolus. Therefore he was compelled to resort to Lamy's scheme, in which he made the injections by means of a catheter passed through the temporal artery into the abdominal aorta. The aorta was compressed both above and below the end of the catheter (laparotomy was resorted to for this) while the injection was made, and thus he was enabled to obtain purely dorsal and lumbar emboli in the spinal cord. The substances used for injection were all sterilized and of the following nature: Lycopodium, pollen of *Typha japonica*, maize, kamala, emulsion of castor oil and atmospheric air.

The examination of the blood system in the dog showed that his arteries were similar to those in man. The territory of the anterior median artery is smaller than in man, as a larger portion of the gray matter is supplied by the lateral arteries.

The pathological examination showed that the reaction of the arterial walls differed according to the cause of the embolus—lycopodium producing the least irritation, while to kamala was due the greatest. This reaction showed itself in a thickening of, and a small-celled infiltration in, the vessel walls. The arteritis was developed as early as the

second day. On either side of the embolus was a dilatation of the lumen of the vessel. Beyond the embolus on its distal side there occurred first an absence of blood due to the vasomotor muscles, which was followed by hemorrhagic infarct. Necrosis of tissue became evident after 29 hours. Cavities were formed and granulation tissue was produced from the connective tissue cells, leucocytes, etc. The typical large granulation cells were of connective tissue origin and persisted longest, being found after some weeks at the divisions of the blood-vessels. The presence of granulation tissue, Hoche avers, permits of no other conclusion than that it was there for the purpose of removing the products of decay and degeneration. JELLIFFE.

- 85 NEUROFIBROMATOSE GÉNÉRALISÉE (Generalized Neurofibrosis). Autopsy. Pierre Marie and A. Couvelaire. (*Nouvelle Iconographie de la Salpêtrière*, January and February, 1900, 13th Year, No. I, p. 26).

This paper is the result of a microscopical study of the tissues of an individual affected with a generalized neurofibrosis in the form of scattered tumors about the size of a pea all over the surface of his body. These tumors are of a fibrous consistency, some show a tendency to become pedunculated. There is no discoloration of the skin covering the tumors; on other portions of the body pigmented areas were found. There was an alteration of cutaneous sensibility all over the body. In addition to the skin manifestations a progressive change in the shape of the thorax was noted. The vertebral column was scoliotic and a gibbosity developed. Patient was very apathetic, which condition progressively increased. Autopsy: The bones of the extremities show no changes; the bones of the thorax and vertebral column are pathologically altered in a remarkable way; the bones are softer and much lighter than normal. In addition to the localized changes, the thorax is asymmetrical and flattened antero-posteriorly. In the small intestine numerous small tumors, similar to those of the skin, were found. These tumors spring from the mucosa. The stomach shows the same condition. Sections from the various nerve trunks show small fibrômas along the fibers. To be noted in this case, from an anatomical point of view, are the peculiar lesions of the bones and the muscles, the fibrômas of the intestine and skin, which developed away from the nerves themselves. The bone lesions suggest the idea of a localized osteomalacia. The origin of these fibrous tumors is still a question of considerable doubt. Recklinghausen believes in the unity of all polyfibromatous growths, reckoning them under the head of a generalized neurofibrosis. There is considerable difference of opinion about this theory; cases have been published by Lahman, Marie, and others, in which the process does not seem to take its origin from the nerves of the skin alone. The authors of this article are inclined to believe that, together with the neurofibrosis, there is also a cutaneous fibrosis, which appears to be developed independent of the nerve branches, namely, in the periglandular connective tissue. It is more the identity of the structure than its origin which makes the term generalized neurofibrosis a fitting one.

SCHWAR.

## Book Reviews.

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THE INTERNATIONAL TEXT-BOOK OF SURGERY BY AMERICAN AND BRITISH AUTHORS. Edited by J. Collins Warren, M.D., LL.D., and A. Pearce Gould, M.S., F.R.C.S. W. B. Saunders, Philadelphia, Publisher.

In the "International Text Book of Surgery," by American and British authors, edited by J. Collins Warren, M.D., LL.D., and A. Pearce Gould, M.S., F.R.C.S., we have a work that will certainly appeal to every surgeon throughout the country. It consists of two volumes. Each volume has about 1,000 pages, with a total of 929 illustrations in the text and 17 full-page plates in colors. The illustrations are beautifully executed and the full page colored plates are truly works of art.

The work represents the labor of fifty-five contributors, nearly every one of whom has a world-wide reputation. The names of the editors are a sufficient guarantee of the high quality of the work.

Volume I deals with the principles of general surgery and general surgical diseases. It commences with a chapter on surgical bacteriology, giving the methods of cultivation, procedure in examination, etc. of the different bacteria causing surgical diseases.

Considerable emphasis is laid upon the importance of the examination of the blood in cases of septic infection, and it is shown how there is an increased leucocytosis in these infections, except in cases of a very mild or a very rapidly running infection.

In the chapter on technic of aseptic surgery, the latest and best methods of sterilization are fully described, not only of the hands of the operator and the site of operation, but also the different materials used for ligatures, sutures and dressings.

Under operative and plastic surgery we have ligation of arteries, amputations, excision of joints, etc., fully described and an easy comprehension of the different steps is afforded the reader from the numerous illustrations. The lines of incision are clearly marked out, so that at a glance, the operation is firmly fixed in the mind of the reader. The subject of fractures and dislocations has received careful attention, and the numerous skiagraphs give us an accurate knowledge of the different deformities produced.

The surgery of the peripheral nerves is devoted to nerve suturing, nerve grafting and nerve stretching, also to a description of the nerve tumors. It shows how certain paralyses may be relieved by surgical interference, *e. g.*, when the musculospiral is compressed by callus formation after fracture of the humerus. The surgical treatment of the different neuralgias, notably trifacial, where the different divisions are destroyed, or, failing in relief, the removal of the Gasserian ganglion.

In volume II considerable space has been devoted to the surgery of the breast and the operation recommended for carcinoma is that devised by Halsted, involving removal of the mammary gland and paramammary tissue, with superficial lymphatics, the sternal portion of the pectoralis major and minor, and thorough cleaning out of the axilla and posterior cervical triangle, if involved. This operation has given the best results of any practised at the present day.

The whole subject of abdominal surgery, which occupies such a prominent position in the surgical world of to-day, comprises the technic of abdominal surgery, peritonitis and a description of the surgery of the stomach, intestines, vermiform appendix, liver, pancreas and gall bladder. The use of rubber gloves is advocated for the oper-

ator. These can be boiled with the instruments so that danger of infection from the hands is eliminated. The different operations performed upon the stomach are described, as well as operations upon the intestines; viz., intestinal anastomosis, enterorrhaphy, etc.

In the chapter on the vermiform appendix, 5 varieties of appendicitis are mentioned, viz., catarrhal, suppurative, perforative, gangrenous and chronic. Wherever an attack has occurred, the appendix is damaged and leaves the person very liable to subsequent attacks. Operative interference is advised and the time of operation should be between the attacks when possible, while the disease is quiescent. This gives the patient the best chance, the mortality with some operators being less than one per cent. Of the different operations for hernia, that of Bassini is recommended as giving the best results, both for inguinal and femoral. Three chapters are devoted to gynecological surgery, comprising a description of the diseases of the vulva, uterus and appendages, and the operations in most common use.

There is a chapter devoted to military surgery, also one to naval surgery, and the wounds produced by modern firearms are described, examples being taken from our war with Spain, and also the British war in South Africa. The chapter on traumatic neuroses is very well written, and will be very much appreciated by many of us who are called upon for expert testimony.

There is one criticism that we do feel called upon to make, and that is that in a work representing the breadth and scope this does, so little space has been given to brain surgery, which is claiming the attention of so many of our best operators and neurologists of the present day. On the whole, it is probably the best work on general surgery we have had placed before us.

C. F. ADAMS.

**RAYNAUD'S DISEASE. ITS HISTORY, CAUSES, SYMPTOMS, MORBID RELATIONS, PATHOLOGY, AND TREATMENT.** By Thomas Kirkpatrick Monro, M.A., M.D. James Macelhose & Sons, Glasgow, 1899.

Two excellent treatises on Raynaud's disease, prepared independently of one another, have recently come from the publishers, one by Barlow, in Allbutt's "System of Medicine," and one by Monro. The latter is considered in this review. Monro's work is very thorough; it contains almost all that is now known of Raynaud's disease, but a careful perusal of its pages leaves the reader with many a question unanswered. The disease is rare. From the statistics of the Glasgow Royal Infirmary it appears that about one case in three thousand admissions to the medical wards represents its frequency, and yet this, probably, is an underestimate. The statistics published in the "Transactions of the American Dermatological Association" show that scarcely one case in 12,000 is found.

Monro points out the resemblance of chilblain to Raynaud's disease. Both are expressions of an undue susceptibility to cold, the former on the part of the tissues, and the latter on the part of the vasomotor system, and cold is one of the most potent agents in inducing these diseases; but in those who are very susceptible chilblain may be caused by a breeze in summer, and Raynaud's disease may be caused by trifling exposure, or may appear as a pure neurosis. Monro finds that in nearly eight per cent. of the cases of Raynaud's disease there is a history of Raynaud's phenomena in some antecedent or collateral member of the family. The occupation which entails exposure to cold is the one most likely to cause the disease, and we therefore find a greater frequency of the attacks in the cold months.

Mental strain, violent emotion, and malaria are recognized as having etiological relation, but the manner in which malaria produces the symptoms is unknown. Monro says local syncope ("dead fingers") is met with in 50 per cent., and local asphyxia (cyanosis) in 94 per cent.; necrosis of tissue, either slight or serious, in 68 per cent. of cases of Raynaud's disease.

Some study of the pathological changes in the disease has been made in the examination of amputated limbs and in necropsies on the bodies of persons who have died from some complication. There seems to be no anatomical condition whatever, either gross or minute, that can be regarded as peculiar to the disease in question. The theory proposed by Raynaud, the theory of vascular spasm, is the one that seems most satisfactory to Monro.

The treatment is rather of a general character. Exposure to cold should be avoided, nourishing diet should be given, etc. Quinine should be tried, as it succeeds in some cases, but fails in others. Opium also is of value.

The title of Monro's book gives a very good idea of the scope of the work. SPILLER.

A TEXT-BOOK OF DISEASES OF WOMEN. By Charles B. Penrose, M.D., Ph.D., Philadelphia. Third edition revised. W. B. Saunders, Publisher.

In this third edition revised, of "A Text Book of Diseases of Women," by Dr. Penrose, we have the subject brought right up to date and placed before us in a very attractive manner.

The author has refrained from going deeply into the special anatomy of the parts, allowing the student to gain that knowledge from general works. Likewise with pathology, he has mentioned only so much as to make the subject clear and comprehensive.

The different diseases of the external genitals, vagina and pelvic organs are described and method of treatment indicated, as well as all the operative procedures that are in most common use at the present day. There is a very good description of Emmet's method of repairing the posterior vaginal wall. This operation, as described by many writers, has been very confusing to the student, but Dr. Penrose, with the aid of his illustrations, has made it perfectly clear.

The spirit of conservatism is apparent throughout the work, and it is just this feature which makes it specially adapted for the use of the student and as a ready reference book for the general practitioner. The chapter on the technique of gynecological operations is particularly clear and to the point; much stress being laid upon the preparation of the patient, the sterilization of hands, instruments and dressings, and the subsequent treatment of the patient. The book itself is a volume of 530 pages, profusely illustrated and is a fine specimen of the bookmaker's art.

C. F. ADAMS.

THE  
Journal  
OF  
Nervous and Mental Disease.

Original Articles.

AUTOPSY IN A CASE OF ADIPOSIS DOLOROSA, WITH  
MICROSCOPICAL EXAMINATION.\*

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The following record is of interest, not only because it is the first autopsy of a case of adiposis dolorosa, with microscopical examination, but especially because it is an autopsy made upon the original case,<sup>1</sup> first published by the writer in 1888. Some two years after this case was described, a similar case was placed upon record by Dr. Frederick P. Henry,<sup>2</sup> and in 1892 the writer grouped these two cases with a third, which he likewise discovered in the wards of the Philadelphia Hospital, and gave to the disease the name of adiposis dolorosa.<sup>3</sup> In 1895, Ewald,<sup>4</sup> described a case which was evidently the same affection, and which he placed in the same category as the cases placed on record by Henry and myself. Subsequently, three cases were described by Spiller,<sup>5</sup> two by Eshner,<sup>6</sup> and one

\*Read at the twenty-sixth annual meeting of the American Neurological Association, May 1, 2 and 3, 1900.

<sup>1</sup>University Medical Magazine, December, 1888, p. 140.

<sup>2</sup>JOURNAL OF NERVOUS AND MENTAL DISEASE, March, 1891.

<sup>3</sup>American Journal of the Medical Sciences, November, 1892. Adiposis to be etymologically correct should of course be written adipositas, yet adiposis seems to be sanctioned by general usage.

<sup>4</sup>Ewald, Berliner klinische Wochenschrift, January 21, 1895, page 58.

<sup>5</sup>Medical News, February 26, 1898, p. 268.

<sup>6</sup>The Journal of the American Medical Association, November 12, 1898, p. 1156.

by Giudiceandrea.<sup>7</sup> Dr. Collins<sup>8</sup> also refers to six cases which have been studied by Peterson and Loveland.

The clinical history of the case here reported has already been published in full. It is briefly summarized as follows:

The patient was a woman, who, when first examined, was 51 years of age. She was a widow, a native of Ireland, and a domestic. The family history presented nothing striking or peculiar. This was also true of her personal history, save that the menopause set in abruptly at thirty-five. Notwithstanding this early cessation of menstruation, she continued to be well until forty-eight or forty-nine years of age. Up to this time she had undergone some general increase in weight, but did not present any of the peculiar symptoms which were noticed later on. A venereal history was denied, but there was probably occasional alcoholic excess.

She first noticed that her arms began to enlarge, and this enlargement was attended by severe pain, shooting and burning in character. It involved both shoulders, the arms, the back and the sides of the chest. It was most marked in the upper arms and back, forming here huge and somewhat pendulous masses. It was elastic, and yet comparatively firm to the touch, and it was impossible to produce pitting. In some situations it felt as though finely lobulated, and in others, especially on the inside of the arms, as though the flesh were filled with bundles of worms. The skin was not thickened; it did not take part in the swelling, and it was not adherent to the subjacent tissues. In addition the swelling was very painful to pressure. Pronounced pressure appeared to be absolutely unbearable. The nerve trunks also were somewhat painful, but this condition was not as marked in them as in the swollen tissue.

The muscles were not involved in the swelling. The affected parts were, however, quite weak. Examined electrically, the muscles of the shoulder and arms yielded a negative result, partly because of the great resistance caused by the intervening tissue. Slight qualitative and quantitative changes were noted in the muscles of the forearms, while in the hands distinct reaction of degeneration was noted in the thenar and hypothenar groups.

Cutaneous sensibility was diminished in various irregular areas over the right arm on both the inner and outer aspects. In the left arm, some impairment of sensation was detected on

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<sup>7</sup>"Un cas d'adipose douloureuse, maladie de Dercum," *Revue Neurologique*, 7, 1899, p. 877.

<sup>8</sup>"Text-Book on Nervous Diseases," edited by Dercum, 1895, p. 898.



the outer aspect of the forearm. Sensibility to heat and cold appeared to have been lessened.

In examining the legs it was found that cutaneous sensibility was distinctly lessened on the right, while showing little or no impairment on the left. There was no alteration of the gait, but both knee-jerks were lost. She complained of a "velvety feel" in the soles of both feet and also in the tips of the fingers.

No enlargement was noted at first in any part of the body, save in the arms and shoulders. There was never any involvement of the face, hands or feet. The face was pale, as were also the mucous membranes. There was, however, a little color in the cheeks, more noticeable at times. Her features were well formed and intelligent. Her hair was dark and fine. Her mind was unimpaired, except that at times she was much abstracted. Sometimes she gave conflicting answers to questions, so that the latter had often to be repeated. The skin seemed dry; sweating appeared to be diminished. There was also some tendency to subnormal temperature during the earlier period of the disease. Her temperature was frequently below 98 deg., and on one occasion as low as 97 deg. After the full evolution of the symptoms, however, this tendency to slightly subnormal temperature was no longer observed.

An examination of the eyes by Dr. de Schweinitz revealed nothing abnormal save some contraction of the visual fields. The other special senses appeared to be somewhat obtunded. An analysis of the urine yielded a negative result. This was also the result of an examination of the blood; there was no leucocytosis.

The patient was under more or less continuous observation at the hospital for a period of eleven years. During this time the neuritic pain, together with increasing enlargement of various parts of the body, formed the striking features of the case. Occasionally small nodules, which had not been discovered before, would make their appearance in various situations. The enlargement gradually involved not only the arms, shoulders and back, but also the abdomen, the hips, the thighs and the legs immediately below the knees. The lower legs, like the forearms, were not affected. The face, hands and feet likewise continued to be free from involvement until the last. On numerous occasions excessive exacerbations of the pains occurred, and not infrequently a simultaneous increase here and there of the swelling would be noted, and the swelling would reveal an increased resistance to the fingers, as though local infiltration or induration had occurred. On several occasions,

when the paroxysms of pain were very severe, the patient would be attacked by persistent vomiting, the vomited matter frequently containing blood in small quantities. Vomiting was particularly likely to occur when the paroxysms of pain involved the fatty deposits over the epigastrium. The patient also suffered from frequent attacks of bronchitis, accompanied by dyspnea and blood-stained sputum. Twice she suffered from attacks of herpes zoster, affecting at one time the left arm and left side of the chest, and at another the back. At various times cardiac weakness became marked, and finally led to her death, which occurred March 5, 1899, the diagnosis at the time of death being, in addition to adiposis dolorosa, fatty degeneration of the heart.

The autopsy was held on the next day, and revealed the following conditions:

The body is that of a woman weighing probably three hundred pounds. There is an excessive deposit of fat in the subcutaneous tissue. It is especially marked over the arms, shoulders, back, and to a less extent over the abdomen and thighs. The hair of the pubis is scant, and there is almost none in the axillæ.

In the median line the abdominal fat is 5 cm. in depth. In the epigastric region a small nodule of fat about an inch in diameter is found imbedded in, but separated from, the general subcutaneous fat like an independent lipoma. Several such encapsulated imbedded lipomata were found in various situations; for instance, the back and the right thigh. On opening the abdomen the intestines are well distended with gas; the omentum is filled with fat; the appendix is free, curved and surrounded with fat; the liver extends 2 cm. below the costal margin on the right side; the thorax is well formed; the ribs are well ossified.

Pleural adhesions exist on both sides; the left pleura shows firm adhesions anteriorly and posteriorly. The right pleura shows the same condition as the left.

The parietal layer of the pericardium is covered by a thick layer of fat; the pericardial sac contains 35 cc. of clear straw-colored fluid. The sub-pericardial fat of the heart is very abundant. On opening the right side of the heart, large currant jelly flat clots are found in the right auricle and ventricle. The left side of the heart contains little blood. The heart muscle is friable and of a yellowish color; the wall of the left ventricle measures  $2\frac{1}{2}$  cm.; of the right ventricle  $\frac{1}{2}$  cm. The aortic valves are slightly thickened; the right auriculo-ventricular opening admits three fingers; the left two. The remaining openings and valves are normal.

The left lung on section shows marked edema; no tubercles are found. The pigmentation is slight. The right lung also shows extensive edema. The spleen measures  $17 \times 7\frac{1}{2} \times 2\frac{1}{2}$  cm. The capsule is wrinkled; the pulp is soft and dark red in color.

Left kidney is surrounded by a thick layer of fat. The ureter is single. The kidney measures  $14 \times 6 \times 3$  cm. On the surface and projecting outwards are seen a number of cysts. The capsule strips with difficulty, and the surface is granular. The surface of the kidney is greasy. The cortex is increased,



Fig. 1.—Transverse section of a peripheral nerve found in the subcutaneous fat, showing diminution in the number of nerve fibers and proliferation of the connective tissue.

comprising one-half of the kidney structure. The color of the anterior surface is yellowish red. The cortex is lighter than the medullary structure. The right kidney measures  $13 \times 7 \times 3$  cm., and closely resembles its fellow.

The suprarenals are apparently normal.

The stomach is rather small; walls are not thickened; mucosa shows some post-mortem change.

The pancreas is surrounded by much fat. Section shows no gross change.

The liver measures 29x20x5 cm.; surface is finely granular; edges are irregular and rounded; the knife meets with a good deal of resistance. Color of cut surface is reddish-yellow brown. Interstitial substance well marked. The bile duct is patulous.

The bladder and uterus are normal.

The brain, cord and membranes reveal nothing abnormal.

The thyroid gland is quite small.

The microscopical examination included a study of the

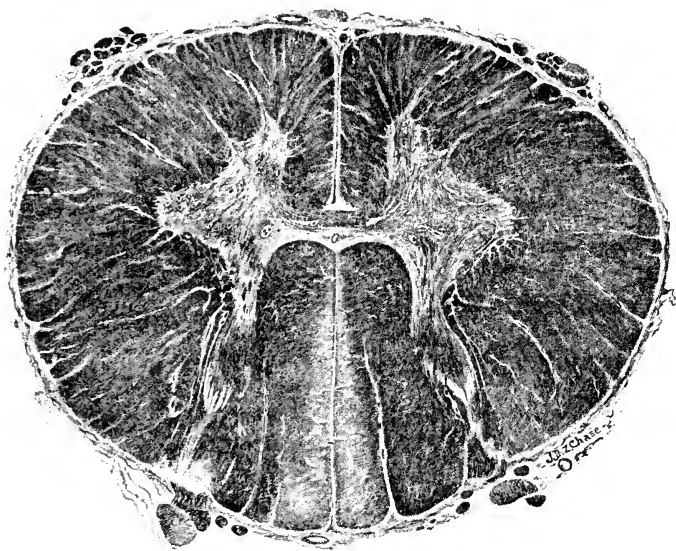


Fig. 2—Section from the upper thoracic region of the spinal cord, showing a moderate degeneration in the columns of Goll.

fatty tissue, the peripheral nerves, the spinal cord and brain, the pituitary body and the thyroid gland.

The fatty tissue in itself presented nothing to distinguish it from ordinary fat. This was not, however, the case with the peripheral nerves found in it. These presented the undoubted evidences of an interstitial neuritis, as will be seen by reference to Figure 1, which shows a transverse section of such a nerve. Here there is seen a marked diminution and atrophy of nerve fibers, together with a marked proliferation of the

perineurium and endoneurium. No changes were observed in the larger nerve trunks.

The spinal cord presents some change in the upper thoracic and lower cervical regions, as is seen by reference to Figure 2. There is a slight degeneration in the columns of Goll. This change is most marked in the upper thoracic region. The lumbar cord fails to reveal any changes.

Various portions of the brain examined reveal no change save unusual pigmentation of the cortical cells.

The pituitary body presents nothing abnormal. The acini and the cells composing them present an absolutely normal appearance.

The thyroid gland proved to be exceedingly interesting. Though not weighed at the time of making the autopsy, it was noted as quite small. The microscopical examination of the gland proved it to be abnormal beyond all doubt. (See Figure 3.) In the study of the sections, I received the kind assistance of my friend, Professor Simon Flexner, of the University of Pennsylvania. Very valuable assistance was also obtained from the paper by Dr. Halsted on the effect of the extirpation of portions of the thyroid gland in the dog in Volume I of the Johns Hopkins Hospital Reports.

A study of the sections reveals the gland to be made up of three or four different kinds of secreting tissue. In the first place, there are large acini distended by colloid material. These large acini vary in size, and their contents vary also in density. The larger acini are globular in shape, while some of the smaller ones are elongated or angular in form. The limits of these acini are clearly indicated by blood vessels which occupy their walls. The epithelium is a single layer, which covers uniformly the peripheries of the acini. Contrasted with these there is another kind of secreting tissue, which is very solid, and in which the acini are made out with great difficulty. They consist of cells filling interspaces of the stroma, and the blood vessels supplying these acini can only be made out in exceptional instances. The lumina of these acini when they can be made out are usually very small. There is here a complete absence of colloid material. In other portions acini are observed which are a transition between the more solid

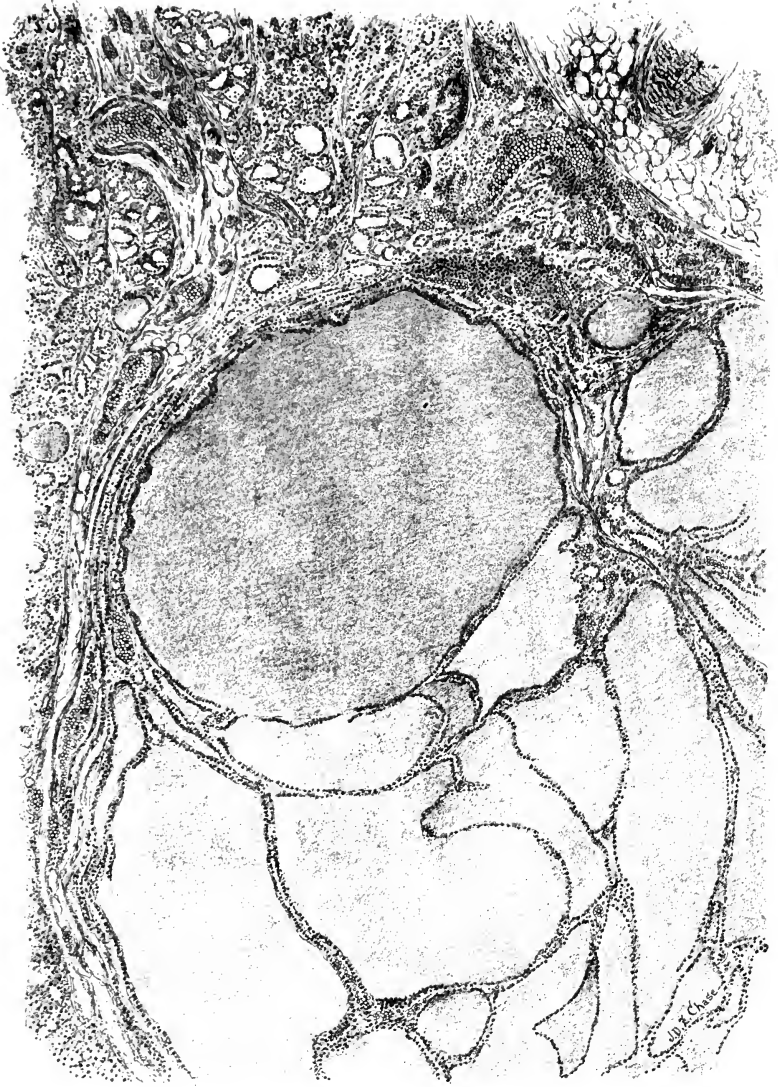


Fig. 3.—Section from one of the lateral lobes of the thyroid gland, showing large acini distended by colloid material. In the central acinus papillary outgrowths of the walls are seen.

nests of cells and the large vesicles which contain the colloid material. In addition, there is a third form of acinus, which is of peculiar interest in that the acini present plications or papillary outgrowths of the walls. These plications or outgrowths project into the lumina of the affected acini, which contain, as a rule, colloid material of lighter staining qualities than the larger vesicles, although not lighter than is contained in some of the smaller vesicles. The epithelium of these last mentioned acini appears at times to be slightly higher than the normal cubical epithelium of the other vesicles. Finally, in some areas, solid masses of cells resembling lymphoid cells are seen, but these are probably young solid acini, like the small acini described above, though the limits of these acini are irregular, because of the absence of preserved blood in the surrounding vessels and of the absence of definite interstitial framework.

The changes observed are indicative in part of hypertrophy. Certainly, this seems to be the only interpretation which can be placed on the numerous small acini which appear to be in process of development. Whether the large acini, distended with more deeply staining colloid material, are to be considered old acini, containing old or altered colloid material, it is, of course, impossible to say, but such an interpretation does not seem improbable. The plications and papillary outgrowths observed in some of the acini are also worthy of comment, in that they evidently represent an attempt to increase the secreting surface of the acini and are again expressive of hypertrophy.

These findings are very surprising, and it is difficult, of course, to frame an explanation. It is not impossible that we have here a hypertrophy which is the direct outcome of a general atrophy of the gland; in other words, a compensatory hypertrophy such as Halsted obtained in the dog after partial extirpation. The gland was small, perhaps sufficiently so to determine compensatory hypertrophy. It is probable, however, that other factors, *e. g.*, qualitative changes of function, also played a rôle in the peculiar symptoms from which this patient suffered. It is not inconceivable that as a result of deranged thyroid action some substance was thrown into the

circulation, which at one and the same time prevented the proper oxidation of the hydrocarbons of the food and tissues, and also acted as a cause of neuritis and nerve degeneration. Whatever the explanation, it is interesting to recall the diminished sweating and the occasional slowness of speech and mental irritability; also observed in that other thyroid disease, myxedema.

It is interesting also in this connection to recall that in the two previous autopsies of *adiposis dolorosa*,<sup>9</sup> the specimens of which, owing to an unfortunate accident, were lost and never microscopically examined, both thyroid glands gave unmistakable evidence of disease. Thus, in one, the thyroid gland was noted as "small, indurated and infiltrated by calcareous matter in both lobes," and in the other as "larger than normal, harder to the feel and much calcified, especially the right lobe."

The writer is indebted to Dr. William G. Spiller for the microscopical work upon the specimens done at the William Pepper Clinical Laboratory.

#### DISCUSSION.

Dr. J. J. Putnam congratulated Dr. Dercum on his very interesting report. He thought that the subject was of importance from another point of view. The question arises whether the disease of the thyroid gland is the cause of the other changes, or whether both occur together. We should not forget the tendency of the thyroid disease to occur as a sign of systemic degeneration. He knew of an instance where a mother and daughter each had a small goiter. The daughter was congenitally blind. A brother showed infantilism and was also congenitally blind and suffered from progressive muscular dystrophy.

Dr. F. X. Dercum said it was unfortunate that we do not know more about the rôle played by the thyroid gland in simple adiposis. It is well known that in this condition beneficial results are often obtained by the administration of thyroid extract, and in *adiposis dolorosa* he had seen treatment by thyroid extract followed by very marked improvement, and in one case a complete cure resulted. It is not impossible that the thyroid gland plays a rôle both in painful adiposis and in simple adiposis.

It was interesting that Dr. Burr had noted changes in the pituitary body as well as in the thyroid gland in a case of

<sup>9</sup>American Journal of the Medical Sciences, November, 1892.



adiposis dolorosa, though this is hardly surprising, knowing what we do of the compensatory relations which appear to exist between the thyroid gland and the pituitary body.

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- 86 AMYOTROPHIE DOUBLE DU TYPE SCAPULO-HUMERAL (Double Amyotrophic Paralysis of the Scapulo-Humeral Type). Georges Guillian (Nouvelle Icon. de la Salpêtrière, Sept., Oct., 1899).

The symptoms in this case developed after traumatism which was unilateral and extraarticular. The case is briefly as follows: A man sixty-three years old, gardener, fell eleven months before in front of a moving carriage. A wheel passed over his right arm. No fracture was found, merely a contusion. Some fifteen days afterward he went back to his work and found that he could raise his right arm with difficulty. There was not any pain and not any swelling. Patient observed on the contrary that his shoulder had grown thinner. This condition grew worse and the left arm became similarly affected.

Examination.—History of heredity or syphilis was absent. The patient presented the typical appearance of a bilateral muscular atrophy of the scapulo-humeral type. Could not raise his arm to a horizontal position, some slight sensory disturbances were present, also some slight pain in the scapular region. Electrical examination showed that faradic excitability was preserved in the trapezius, infraspinatus, rhomboid and deltoid of each side. The contractions were normal. Galvanic excitability was diminished in these muscles. No polar changes observed at all. The tendon reflexes were increased. The further discussion of this case seeks to answer these questions: What is the variety of muscular atrophy present; what is the cause of the condition; what is the anatomical condition, and what is the prognosis? Differential diagnosis had to be made from hysterical paralysis, brachial traumatic paralysis, Erbs' scapulo-humeral myopathy, Aran-Duchenne progressive muscular atrophy, scapulo-humeral type of Vulpian, chronic poliomyelitis, traumatic syringomyelia, pachymeningitis cervicalis hypertrophica and amyotrophic lateral sclerosis. These are all excluded and the condition is believed to be one of bilateral muscular atrophy of peri-articular or articular origin, the so-called reflex type.

The prognosis is not a bad one. Of interest in this case is the bilateral atrophy in spite of the unilateral traumatism. There are a number of such or similar cases reported in literature by Charcot, Raymond and others. The reflex theory of this condition has been proven experimentally by Raymond, who injected nitrate of silver and other irritating substances into the knee joint of animals. Atrophy of the muscles followed. No lesions of the cord have been found in the cases reported. The condition is due to dynamic action and to the irritation of the terminal fillets of the articular nerves.

SCHWAB.

## IMPERATIVE IDEAS IN THE SANE AND THEIR MANAGEMENT.\*

BY EDWARD B. ANGELL,

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"It is a great gift," says Taine, "to know how to forget." Were it possible for the human mind to take cognizance only of that which it chooses, much of the misery of existence might be spared.

Even in the normal mind constantly are recurring words, phrases, emotions or ideas, which force themselves against the will upon the attention and interrupt the logical course of mental activity. But in a morbid state of the mind this domination of imperative ideas becomes a disease, against which the most strenuous efforts of the will seem impotent.

I need not stop to relate cases of this type. They are familiar to all who have aught to do with the management of mental disturbance. While imperative ideas are more pronounced in the insane, yet they frequently torment minds otherwise well balanced, requiring the utmost ingenuity and patience of the physician to dislodge them. "It will be a precious discovery for psychiatry," says Janet, "that will enable us to create forgetfulness at will."

My purpose then in bringing this subject to your attention is to determine how far our present knowledge, through proper classification and analysis of these imperative ideas, will enable us to recognize their significance and to call forth a discussion of the measures that have been found serviceable for their relief.

Unfortunately the psychologists and alienists are not uniform in their interpretation of the term "imperative idea," and it still remains to establish a definite and uniform meaning.

According to Tuke, "Imperative ideas are morbid suggestions or ideas imperiously demanding notice, the patient being painfully conscious of their domination over his wish and will," a condition very different from that produced by the "fixed idea" or delusion of the insane mind. In one case

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\*Read at the twenty-sixth annual meeting of the American Neurological Association, May 1, 2 and 3, 1900.

(imperative idea) the psychical reaction in feeling is against the idea, in the other (fixed idea) it is in harmony with it.

The French phrase, "*Obsession*," corresponding to the German, "*Zwangsvorstellung*," more properly expresses the concept. Obsession includes the idea of antagonism, and thus even more clearly than "imperative idea" defines the conscious struggle of the ego against its acceptance. This conscious and even painful resistance of the mind is an essential element in the condition. Therefore we regard the terms imperative idea and obsession as identical, both involving conscious resistance of the will.

Even Janet, who has done much in this field, is not always clear in his distinction between obsession and *idée fixe*, indeed, he uses the terms interchangeably.

According to Mendel, obsession or the "irresistible conception" of Westphal is not dependent upon affective sensibility; it is an unalterable or pathological association of ideas obtruding itself in the course of a normal ideation, well called by Morselli, "rudimentary paranoia."

Pitres and Régis, in a communication to the Congress at Moscow, took the ground that the emotional element dominates obsession or the imperative idea, while the intellectual dominates the fixed idea or delusion; although in regarding the conduct of patients the affective sphere is really involved in both.

According to Keraval obsessions appear suddenly and involuntarily interfere with the course of ideas of the subject, produce an irruption in consciousness, impose themselves with annoying energy upon the attention while repelling the judgment, and disappear spontaneously. Further, he says, "An obsession is a fixed idea, undoubtedly, but it is recognized for what it is, and generally it takes the form of an attack and is always accompanied with a provocation to resist it, to fight against it on the part of him who feels it—quite different from the unreasoning acceptance of a true fixed idea or delusion."

Such then are the varying definitions given to this term obsession or imperative idea. The consensus of opinion, however, strongly favors a real discrimination between the

imperative idea or obsession and fixed idea or delusion, and a clear comprehension of this distinction will aid materially in clearing up the subject.

Two elements then are indispensable to obsession, to quote Legrain:

"(1) *A center* which suddenly and isolatedly enters into function, its action not being required by the mental needs of the moment;

"(2) *Temporary impotence* of the will to remove this obsession."

In the investigation of these morbid ideas it is necessary to study the mind in its automatic activity, the condition which to a very large extent prevails in states of sleep, hypnosis, somnambulism, hysteria and insanity, during which normal volition is largely or wholly suspended.

Investigations of dream phenomena throw much light upon the subject, and could dreams be recalled in the waking state much aid in diagnosis and treatment could be gained by an analysis of their source and nature. Dream ideas are not corrected by associated ideas of consciousness, which, by the way, has little to do as an agent in the functions of recollection and imagination, and such ideas exist therefore in exaggerated proportion.

Such, too, is the nature of imperative ideas. They are waking dreams, which the feeble will is powerless to eliminate from the troubled field of consciousness.

Dreaming is commonly attributed to digestive disorders, but more properly, I believe, is dependent upon disturbance of any one or more of the elements of somatic consciousness.

The unity of the ego, the sense of personal identity, is largely related to subconscious somatic sensibility or cœnesesthesia. Upon the preservation of normal proportion in its elements depend the feelings of well being. It is in some modification of this subconscious sensibility that a large proportion of imperative ideas take root. That is to say, imperative ideas are largely emotional in their nature and related to abnormal sensations of a somatic character.

This is the common mode of origin of primary imperative ideas, which, indeed, are largely subliminal. They, however,

give rise to secondary obsessions which manifest themselves in the affective or intellectual sphere, and become the morbid phenomena with which we are ordinarily acquainted.

In the normal condition, subjective phenomena of somatic consciousness are largely subliminal. When through disordered function they enter the field of consciousness, they are interpreted objectively in accordance with ordinary sensory experience. Hence it is that our patients give definite interpretations to their feelings. Agoraphobia, for example, is the subjective aspect of a motor instability. The obsession of doubt is related to the loss of the sense of personal identity due to an abeyance of co-ordination of mental function. Hallucinations of movement, *i. e.*, vertigo, due to disordered action of motor centers, are entirely subjective, but are interpreted objectively in accordance with ordinary sensory experience, just as sensations of subjective origin are interpreted objectively and become hallucinations.

Furthermore, imperative ideas are dependent for their manifestation upon a state of mental feebleness, an insufficiency of cerebration, an instability of mental synthesis.

The subjects of this mental vice may be divided then into two great groups; primarily, those whose disturbed mentality is dependent upon hereditary, congenital or acquired defects of organization, resulting in ill-balanced minds—the degenerates; secondarily, those who through accidental causes, education, traumatism, the intoxications, infectious diseases or nervous fatigue, become unduly subject to emotional disturbance—the weak-willed or, to adopt the French, *les abouliés*.

In a general way these ideas may affect either the sensorium, producing uneasy sensations; the motor sphere, causing impulsive acts, or produce vaso-motor disturbances such as blushing, etc.

Aside from inherited or acquired defects of the organism, for which we can do little, the development of imperative ideas is dependent upon certain conditions which Maudsley has well analyzed in a study of the dream state, such as:

- (1) Character and precedent mental experience.
- (2) Impressions on a special sense.

- (3) The state of muscular sensibility.
- (4) Organic or systemic impressions.
- (5) Conditions of cerebral circulation, and
- (6) The state and tone of the nervous system.

Mental deterioration signifies a reversion to the infantile type, hence it is that such minds become more automatic, more the theater of the emotions, fertile soil for the development of these morbid ideas.

Childhood furnishes numerous examples of what we might term normal obsessions, which never become impulses and ultimately fade from the field of consciousness.

In the pathological obsession, however, the victim is painfully conscious of its dominating character, but efforts to thrust it aside only result in mental anguish—*angoisse*, the French authors well name it—and a sense of temporary relief is only secured by yielding to the suggestion or impulse. If it be an impulse to commit a compromising act, further struggle against obeying its demands implicates the body and causes acceleration of the pulse, heart palpitations, precordial distress, an outbreak of perspiration, even tremors and physical pain.

Recession of the field of consciousness is perhaps its most general characteristic. This may go so far as to result in absolute fixation of subjective attention, when, as one patient well complained, "the actual life had become unreal, the dream life or reverie replacing real existence."

The proper treatment, or rather management, of the imperative idea is as yet an unwritten chapter in therapeutics. I am aware that many will say, treat the underlying physical exhaustion or functional perversion. But do you find that fully satisfactory? The body may recover; does the mind always respond? Alas, no; and many who have well recovered their normal physical health limp through life the prey to morbid notions and erratic impulses.

To argue against the reality of such ideas!

"You may as well  
 Forbid the sea for to obey the moon  
 As or by oath remove or counsel shake  
 The fabric of his folly, whose foundation is  
 Piled upon his faith, and will continue  
 The standing of his body."

It is the emotional state and not its representation in consciousness that we should seek to correct. The primary requisite to satisfactory management, then, is a careful study of the nature and manifestation of the imperative idea. Its proper classification will oftentimes furnish a key to satisfactory treatment. If it depends upon the feebleness of mental synthesis, treatment should be directed toward developing the power of attention by education as well as by other measures. If, on the other hand, the imperative ideas are the most important conditions, the source of their origin in somatic consciousness must be determined and so far as possible the cause removed. Oftentimes this disturbance in somatic consciousness is due to a perversion or loss of normal feeling and the morbid idea therefrom can be referred to a peripheral source. Morbid ideas which result from disturbance of this character may often be replaced in the consciousness by the development of real sensation through peripheral stimulation. This is the principle of replacing a morbid idea by a real one, but it is essential for a successful result that normal sensibility be aroused at the point which is the seat of the morbid feeling.

Isolation is another important method in securing control of such disordered mind. By removing the patient from his accustomed environment we do much to bring about a dissociation of the imperative idea. This dissociation also may be furthered by resource to bribes and penalties, whereby the attention is stimulated in a more wholesome direction. Isolation for the feeble-minded is also of value inasmuch as it enables him to escape the numerous though petty demands on his mental existence; but isolation if prolonged itself may fail through the patient becoming too readily accustomed to his new associations. A frequent change is, therefore, of value in breaking up the continuity of these imperative ideas. I am in the habit of breaking up the day for the worst class of cases into small portions, arranging a chart of daily routine that admits of frequent change and variety, to avoid wearying the enfeebled mind by too prolonged attention.

Hypnotism, to which French physicians have resorted so much of late, both for the purpose of investigating this con-

dition and for its proper management, certainly offers a promising means of relief. Through means of the hypnotic state often a knowledge can be obtained of the underlying, primary disturbance which, existing as a subconscious idea in the mind of the subject, otherwise never would come to light. If once this primary disturbance can be discovered the ultimate relief becomes easy. Hypnotism, as Janet well says, is not a rapid treatment, miraculous in character; it is rather a species of education which should endure for some time in order to be successful. It should not be interrupted suddenly, even though in fortunate cases the cure seems satisfactory. The seance should be repeated, but with gradually increasing periods of intermission. It should never be resorted to except with the consent and understanding of the patient, nor should it be employed, for obvious reasons, except in the presence of a third person. Another important feature in the employment of hypnosis is that a patient should always be carefully awakened and left in a calm mood at the expiration of the seance. Undoubtedly through this means some remarkable results in disestablishing the authority of the imperative idea have been secured.

Janet's recent work on "*Neuroses and Fixed Ideas*" presents a very thoroughly studied group of such cases and is well worth the attention of those who are compelled to deal with these conditions. The hypnotic or somnambulistic state, prolonged for hours or days, seems to have a special value in curing well-seated imperative ideas or obsessions, such as hysterical contractures. Hypnosis is only of value in the cases of the emotional type so-called. It has no influence whatever in the non-emotional type, that dependent upon heredity or acquired defects of organization.

Much may be done through a process of education; by mental gymnastics; by efforts at fixing the attention; by arousing in the mind antagonistic ideas, or by association of absurdities with the imperative idea, the principle of dissociation through substitution or diversion. Undoubtedly under similar conditions what has been done for idiots and feeble-minded, through proper training, can be done for the degenerate or the weak-willed. I believe in time that this



method of education will enter largely into the sanatorium treatment of the more unfortunate of this class of cases. In a general way much may be done to lessen emotional influences by training the patient to withstand painful stimulation of the skin. For this purpose nothing serves better than the cold douche, static electricity or the faradic wire brush.

It is needless to say that above all else it is necessary for the physician to secure the confidence of his patient. Oftentimes the revelation on the part of the patient of the nature of his imperative idea and its corresponding disturbance in consciousness will relieve the mental stress and cause it to fade away. In many cases, however, it is necessary to take charge for a long time of the distempered mind, and by constant advice and oversight direct its activities into a normal and healthful channel.

Such are the various measures whereby we may hope to bring relief to those whose life is made miserable by this imperious tyranny.

So far as the measures which may be used against the physical conditions which furnish the fruitful soil for these imperative ideas, I have little to say. They are well known to all. One condition, however, which frequently I have found to underlie these phantoms is lithemia. The relief of this by salicylates, alkalies, and proper diet is often attended with magical results, and it becomes a much easier task to divert the attention from subjective sensations and reduce the exaggerated self-consciousness. It is rather the treatment of the imperative idea itself with which I have concerned myself. If I have but exposed how little is known regarding this feature of psychical disturbance, I trust it may stimulate the members of the Association to closer study and later to more satisfactory results.

## CHRISTIAN PSEUDO-SCIENCE AND PSYCHIATRY.

BY SMITH BAKER, M.D.

### ABSTRACT.

Dr. Baker said that he would like to turn the attention of the Association to certain lines of investigation that seem to promise much if properly carried out. He did not recall instances in which, if he could carry the investigation far enough, he could not find that antecedent to conception there was a deterministic condition of the parental mind, in the form of a stress brought about by deprivation. Take, for instance, cases of sexual obsession. So many times he had found that one of the parents, or both of them, had lived a life of absolute sexual stress, of the order of deprivation. He would not go into the details, but simply spoke of the general fact to indicate a line of investigation. Take, again, parents who, during their child-bearing period, are eager for property and yet, who, to a certain extent, are deprived of the benefits of property. Such a stress of mind, or emotion, or body, if you please, seems to be the ground-field in which the child gets the predisposition to an imperative conception or obsession, which will be developed later on. He did not say it is so universally; but he did say that when he had an opportunity of carrying the investigation far enough, he could not recall an instance where he had not struck the dire influence of stress upon determining the predisposition of the child.

With reference to the influence of hypnosis in the treatment of these conditions, he confessed that he was not enough of a hypnotist to bring about the result claimed. He would rather take ten times the pains to engage such people in a straightforward development of the mind, by putting them at Emerson's essays, or geometry, or some new science; by giving them a hold upon something new to work with, than to use all the hypnosis that he was capable of bringing about.

### DISCUSSION.

Dr. Philip Coombs Knapp said he hardly thought that the condition which Dr. Angell had made of imperative conception and fixed idea is one that can be strictly maintained in practice, for it certainly had been his experience to have noticed almost every stage between the mere obsession of the healthy

person, as for example, when some tune or verse of poetry gets fixed in the mind for a period of six to twenty-four hours up to the quite profound obsession, gradually developing into a delusion. There can be no sharp lines drawn between them.

In regard to treatment by hypnosis, although Janet admits that individual fixed ideas in his patients may be removed by hypnosis, still he recognizes that the tendency persists and that such persons are very susceptible to new fixed ideas. Attempting to remove the fixed idea by hypnosis seemed to Dr. Knapp something like trying to cure a weak back by putting on a spinal brace. By instructing the patient as to the nature or unreasonableness of his idea, it encourages him to correct it and to overcome it, and thus he can be cured more efficiently and more permanently. A typical example of that is the case of Goethe, who, in his early years, was a victim of the fear of high places, and who cured himself by exposing himself to danger on the spire of Strasburg cathedral.

Dr. S. Weir Mitchell desired to call attention to one or two points of interest that occurred to him in regard to these matters while his friend and former pupil, Dr. Angell, was speaking.

A lady whom he had recently seen is possessed of a habit which is not absolutely imperative. A good many of these obsessions are not so imperative as to be totally and entirely beyond control, and there is every possible amount of difference as regards the matter of self-control. If it were not so, if there were no margin on which we could stand to help them, there would be no possibility of assisting many of these cases. This lady had a mania of repetition in one of its most interesting forms. Whenever she did certain things, like moving a book on the table, she felt obliged to move that book a certain number of times, and associated the number of times she moved the book with some one of her relations, there being a numeral in her mind for each particular relation. This had become so serious a matter that if she once turned a book on the table she must turn it three times, if thinking of her cousin; and seven times if she were thinking of her husband. This became worse and worse until it attracted attention. Dr. Mitchell asked her what was her feeling about it. She said: "When I was a child I had a tic"—what we call "habit spasm," if you like—"and if I did not obey the impulse to move my two shoulders I was so uncomfortable that I yielded to it, and I have the same kind of a feeling in regard to this." She said: "If I do not turn the book three times when I think of my cousin, than I have a discomfort of mind that is comparable to the kind of discomfort I would have when I did not obey the

impulse to move my shoulders as a child, and I was old enough to remember it." Dr. Mitchell said it was a sort of mental tic. He thought her illustration exceedingly apt and interesting. He asked whether she were getting better. She said yes. "A cousin of mine has been with me and has found a remedy that helped me. Whenever I do this thing she pinches me and I have got so accustomed to associate the discomfort of the pinch with the tendency to turn the book around, but though now I am getting rid of it, when she doesn't pinch me there still comes to my mind the disagreeable idea."

Dr. Mitchell desired to say a word in regard to what Dr. Baker had spoken about. Dr. Mitchell had had a very long and large experience in hypnosis. It began in his earlier life and he had used hypnosis longer than anybody here. He did not recall a single case—and he was speaking with great gravity and thought of a serious nature—which had been cured by the simple use of hypnosis alone—he meant a permanent cure. He was glad to have the support of Dr. Baker as to the value of more logical and certainly more wholesome methods of dealing with people, and he was inclined to use these rather than hypnotic suggestion. Dr. Mitchell believed that hypnosis will practically disappear in the practice of medicine and pass into the hands of persons who alone will use it and use nothing else.

Dr. B. Sachs thought that the subject of imperative concept is one which interests us, because it is a practical, psychical phenomenon, which very closely comes to a physiological condition and does occur in persons who are otherwise normal. He said they are otherwise normal, for he confessed that as a boy he was subject to an imperative concept, and an experience which he had always interested him as proving that these concepts do not start up in the mind quite as spontaneously as is generally supposed, but that the imperative concepts are often based on some definite reasoning. When he was a boy of seven years he was unfortunate enough to lose one parent, and the emotion attending the loss of that parent suggested to him that it would be more unfortunate to lose the second parent. With that idea the number two gained such an influence over his mind that from that time on for several years he dreaded doing anything twice or in multiples of two. He would not do a thing twice or four times. He would do it three or five or seven times. With that idea in mind other imperative acts followed very readily. For instance, before going to bed he would like to make certain that he did not touch the floor twice, that he touched it only once or three times. Consequently, he was in a state of uneasiness and

would get out of bed in order to make sure that he only touched the floor three, or five, or seven times. He was cured of this by a brother, who thought that Dr. Sachs' actions were nonsensical and who persuaded him it was just as well to get rid of these concepts, and once rid of them they have never troubled him since.

As regards the effect of hypnotism, Dr. Sachs was very glad that Dr. Mitchell had spoken as he did. An experience that Dr. Sachs had had within the last week or two proves that far from curing imperative concepts, hypnotism may be responsible for their origin. The patient was suffering from hypochondria and neurasthenia. She was entrapped by a hypnotist, who thought he could rid her of this condition by making her repeat three words. She is now constantly repeating these three words and is much annoyed.

If Dr. Mitchell said that he had never seen a cure by hypnotism in these cases, Dr. Sachs thought we can add there is even some danger lurking in it as originating the imperative concept.

Dr. J. J. Putnam said that Dr. Sachs' remarks recalled an interesting case reported by Janet in one of his recent books. In this case Janet describes a patient who had been terribly alarmed at the idea of taking cholera, with which her husband had died, and carried this dread constantly with her, saying "cholera" over and over to herself, until, as he thought, the fear had become associated rather with the word than with the idea. He conceived the plan of substituting for the word "cholera" others of similar sounds, but different meaning, as "choler," "color," "coal," etc., and in that way, with a little adroit maneuvering, he cured her of that particular trouble.

As to the matter of hypnotism, Dr. Putnam had a certain loyalty to the method for the good that it has made possible. It seemed to him that the idea has often been misconceived. He thought there was no difference between the use of the hypnotic methods, strictly speaking, and the various other methods by which we take advantage of temporary passive states in our patients to influence them in various ways for their benefit. Probably we should all agree that when it comes to the cases such as severe sexual perversions reported by Lloyd Tuckey, where the patient's will is almost helpless, and to some of the drug-habit cases, the use of hypnotism has proved of real value. Dr. Putnam had not used hypnotism, in a strict sense, very much, but he had frequently recommended its use and had recently been treating, through an assistant, a boy with incontinence of urine and feces, with excellent result.

Dr. Smith Baker remarked that he was always happy when he could get his patients to actively respond to something. The passive condition into which they drift he is afraid of. The active condition, in which there will be response to something, is one that gave him satisfaction.

Dr. E. B. Angell said that this subject is one that we have to deal with in our daily work, and he confessed that he was more anxious to know how to manage satisfactorily these cases than those of any other class. He believed that if we study these cases carefully and analyze their imperative ideas, we shall find some way to counteract these ideas. It is a shame to American as well as to English neurology, that we have not done as much in this line as the French. He knew of very little, aside from what Dr. Mitchell had done, in regard to methods that are directed against these ideas themselves.

So far as Dr. Knapp's suggestion was concerned, he thoroughly recognized the difficulty of establishing any definite or absolute distinction between the fixed and imperative ideas. Of course, these conditions are largely relative, but he did believe this distinction between the fixed idea and the imperative idea is possible. The imperative idea is attended with antagonistic feeling in the consciousness of the patient, a strong desire to resist the dominance of the impulse. As an illustration there occurred to him a recent patient who was a masturbator. This patient did not want to masturbate. There was no pleasure in it, but when he went to bed he was so afraid that he would do it that the fear itself led to the act, effort to avoid it only resulting in a wakeful nightmare. Such is obsession or an imperative idea. This man was compelled to do this act, not for physical satisfaction, but from psychical pain. We know what the fixed idea is; a real delusion, which is in full harmony with the patient's feelings, however unreasoning his conclusions. If we bear these distinctions in mind they will help us. The imperative idea we can do something for; the fixed idea we cannot.

Dr. Angell said he had introduced the subject of hypnotism because, through it, we find out a great many things about the imperative idea. Janet regards it as a means of education, and a more satisfactory method than education in the waking state. However, Chaslin recently informed Dr. Angell that they are not using hypnotism at the Salpêtrière so freely since the death of Charcot. In some cases, however, it may be an efficient method for educating patients who are dominated by an imperative idea.

Dr. Angell did not believe that American men and women

are as susceptible to hypnosis as are the French, or Germans, or other Continental races.

With reference to the delirium of doubt, referred to by Dr. Sachs, that is pretty generally supposed to be due to a disturbance of the sense of personal identity due to abeyance of the co-ordination of mental function. Dr. Angell believed that in the future, by trying to find the basis upon which these ideas rest in the sub-conscious mind, we can do something more than we have done in the past for their removal.

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#### 87 ERUCTATION, REGURGITATION AND RUMINATION.

Although these conditions may be associated with organic lesions of the stomach, they are usually the result of functional disturbances or are "habit neuroses," and it is this latter phase which is reviewed by H. W. Lincoln (*N. Y. Med. Jour.*, March 24, 1900). Eructation consists, at first, of the belching of gas, later, of air which has been previously swallowed. No doubt at first there was more or less gaseous distention, and as eructation brought relief, the habit was formed. There may also be esophageal eructation, in which the air never reaches the stomach. In treatment, suggestion is all important. The patient must make every effort to suppress belching. Bromides may sometimes be of aid. Regurgitation usually comes on during the stage of digestion, especially just at the close of a meal. If it comes on later the taste varies; an hour after eating the taste is sour, due to butyric or lactic acid, or intensely sour and corroding due to hydrochloric acid, or perhaps bitter, due to peptones. It is believed by the author that atony is a prominent factor in these cases. It occurs, of course, in stagnation, cancerous conditions, etc., but the malady is usually of a nervous origin. The patient should always be forced to swallow the food again as fast as regurgitated, and to eat always in company and slowly. The diet should be arranged according to the gastric secretion, and should be light and nutritious. Electricity externally and internally may do good. Small ice pellets are recommended by Alt. Rumination is more rare and may be continued for years with impunity. Heredity, custom and imitation stand pre-eminent as causative factors. It is a motor dynamic affection of the stomach, occurring among all classes, frequently among idiots and the insane. It is confined to the period of digestion and not accompanied by any symptoms of discomfort. The chemical condition varies from achylic to hyperchloridic. The treatment for regurgitation given above should be strictly enforced, and the patient should be warned against the contraction of the abdominal muscles. A good idea is to administer at meal-time some extremely bitter preparation, as quinine or a combination of condurango, quassia, gentian, nux vomica and capsicum. A patient usually hesitates before regurgitating this mixture a second time.

JELLIFFE.

## A CASE OF MONOCULAR HYSTERICAL AMAUROSIS IN A GIRL ELEVEN YEARS OF AGE.\*

By C. A. VEASEY, M.D.,

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Hysterical amaurosis in a child seems to be of sufficiently infrequent occurrence to permit of the report of the following isolated case.

On the 5th day of January of this year, E. C., a girl eleven years of age, was brought to me for an examination of her eyes, with the history of total blindness in the left of one week's duration. For several weeks preceding the attack of blindness she had complained of supra-orbital and retro-ocular pain on that side to which but little attention was given by her parents, as she had always been very healthy and as there were no external evidences of any disease. One morning, without any unusual attack of pain, or any premonition whatever on the preceding day or evening, she awoke stating that she could see nothing with the left eye. But little attention was paid to the blindness for several days, the parents believing it was some temporary matter that would disappear, but as the condition continued she was taken to the family physician, Dr. W. G. McNaul, who at once referred her to me for examination.

Upon making the usual tests the patient read 6-6 with the right eye, but with the left could not distinguish concentrated light. There was no external evidence of any disease and no pain upon pressure of the eyeball or surrounding parts. The ophthalmoscope revealed a perfectly healthy looking fundus. The pupillary reaction was normal. There was some anesthesia of the cornea and surrounding conjunctiva, and this causing me to think that possibly hysteria might be the origin of the trouble, Harlan's test was employed, when the patient readily read 6-6 with the so-called "blind eye."

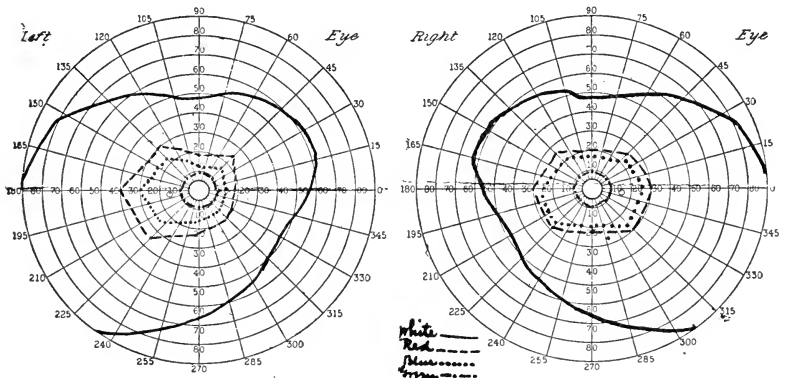
Before allowing either the patient or the mother, who was sitting in the office, to discover that the sight was present in the left eye, the fields of vision were taken, with the results as

\*Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, April 17, 1900.



shown in the accompanying charts. There is marked contraction of the color fields, with complete reversion of the red and blue fields, that for form remaining normal.

Even during the taking of the fields the child apparently did not discover that she was seeing with the left eye, though the right was necessarily covered. There were no areas of anesthesia on the face, excepting the cornea and conjunctiva, and I now regret that I did not have an opportunity to test for them in other portions of the body, but this was not done at the time. The patient was a child of good disposition, fond of going to school, not given to attacks of ill temper, and not more emotional than was usual for her age. Menstruation had not been established.



The compound syrup of hypophosphites was ordered, and she was assured that it always cured cases like hers in a very short time, and on the following day it was ascertained that the vision had remained from the time of its re-establishment during my examination. I have seen her within a few days, and though her vision is normal in each eye, and there is no anesthesia of the cornea or conjunctiva, the color fields are still somewhat contracted and reversed, as at the first examination.

Naturally, it may be suggested that the child was malingerer, but I think that her age, her disposition, the anesthesia of the cornea and conjunctiva, the reversal of the color fields, and the fact that no effort was made to conceal the vision after it had been re-established, are against this suggestion.

As to the method of re-establishment of vision, I can only quote what has so frequently been said in connection with this class of cases. The eye probably sees all the time, but the effort is not properly recorded until something causes the so-called blind eye to assume the burden of the visual act. After the vision has been re-established in this manner it remains for a longer or shorter period, depending upon the nature and intensity of the hysteria.

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- 88 THE EVOLUTION OF MODESTY. Havelock Ellis (*Psychological Review*, VI, 1899, p. 134).

The distinguished philosopher of sex here defines modesty provisionally as "an almost instinctive fear, prompting to concealment, and usually centering around the sexual nature," two fears especially combining in its biological production, one, the female's actual "fear" of the male as an aggressor, the other, more distinctly human and social in its origin, the fear of causing disgust. Another factor of modesty, particularly among savage races, both ages ago and at present, is the idea of ceremonial uncleanness. Dr. Ellis thinks, after Westermarch, that clothing originated less to conceal than to give prominence, and the continuance of its use was helped by the apparent protection of the property rights which a husband has in his wife afforded by clothing—a presumption not too well based, as it seems.

Blushing or at least the possibility of blushing, Dr. Ellis considers, causes the modesty more properly than does modesty cause the blush. Darkness tends to repress modesty by lessening the probability of causing disgust. The often reported fact that many peoples deem it the height of immodesty to eat before witnesses is accounted for here by the presupposition that in early times when food was hard to obtain the sight of one eating would naturally arouse "a profound emotion of anger and disgust to see another person putting into his stomach what one might as well have put into one's own"; as kindness developed this would have led to seclusion when eating. It is not at all obvious how this explains the original "disgust," however natural the postulated anger.

While civilization expands the range of modesty, it renders it more capricious and changeable, and, thinks the author, on the whole, less prominent. "Modesty is a part of self-respect, but in the fully developed human being self respect itself holds in check any excessive modesty." The tendency of civilized life is to render modesty "a grace of life rather than a fundamental social law of life." DEARBORN.

- 89 POLYNEURITE TUBERCULEUSE DOLOREUSE (On Painful Tubercular Polyneuritis). H. Du Four (*Revue Neurologique*, Feb. 15, 1900).

The history is given of a woman thirty years of age with previous alcoholic and tubercular history, with enlarged cervical glands. She had extremely painful sensations in the muscles of the lower extremities. The reflexes were increased. There were no motor signs and the electrical reactions were normal. Diagnosis was arrived at by exclusion. JELLIFFE.

ON THE USE OF A NEW, SAFE, AND EFFICIENT HYPNOTIC IN THE TREATMENT OF THE  
INSANE: CHLORETONE.

By J. PERCY WADE, M.D.,

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Hypnotics are certainly the most used of any class of remedies in the treatment of the insane. A hypnotic answering the description in the above title would surely be a source of gratification to those having charge of this most unfortunate class of the sick.

Chloretone is described as a white, crystalline powder, formed through the interaction of chloroform, acetone and an alkali. It has a strong odor of camphor, is only partly soluble in water, but is soluble in alcohol. When placed upon the tongue it first produces a stinging sensation, which rapidly gives way to an anesthetic condition.

It is claimed by those who have experimented with the drug in the laboratory that it is not a depressant to the circulatory system. The pulse rate is slightly decreased, but the action of the heart is not disturbed. The blood pressure remains unaffected, and the respiration shows no change. Locally it is an anesthetic, and a solution of it when applied to any part produces a local anesthetic condition in a short time. Given internally it is said to paralyze the sensory nerves and produce a general anesthesia.

Although no records have been kept in this hospital as to the action of the drug upon the system, yet the general observations agree in the main with the above deductions. It was found to reduce the pulse rate, but not to decrease the heart action. The arterial tension and the respiration were apparently unaffected. The secretions seem to remain unchanged. The drug possesses considerable power as an anesthetic, when given internally, and this fact was demonstrated in a case of acute mania, with an extensive cellulitis of both forearms. Both the arms were freely incised while the patient was under the influence of the drug, apparently without the production of any great amount of pain. After the patient had recovered from the attack, and upon questioning,

she stated that the operation did not cause her any pain. In no case did the drug produce any irritation of the stomach, nor were there any bad after-effects complained of by the patients for whom the drug was prescribed.

Chloretone is but slightly (0.8 per cent.) soluble in water, but is soluble in alcohol, and the best vehicle in which to administer it was found to be sherry wine or whiskey, or it may be suspended in syrup of acacia, or given in tablet form. Where the patient refuses to take food or medicine, it can be given in milk through the stomach tube at such times as the patient may be fed.

The ordinary dose of chloretone which is recommended to produce sleep is from 6 to 18 grains, but 6 grains are absolutely too small and are useless in the treatment of the insane. The initial dose should be at least 15 grains and should be gradually increased until the desired effect is produced. The average dose given at this hospital was about 35 grains. Some of the patients exhibited susceptibility to small doses of the drug, and in a few as low as 20 grains affected them favorably. When necessary to produce an effect as much as 50 grains was given without any unfavorable results. The larger doses should not be repeated the next day, unless absolute stupidity is desired.

The drug was employed as a sedative and hypnotic in eighteen different cases, 11 women and 7 men. These represented many and varied forms of mental disorder. Four of these were cases of acute mania of recent duration, in two of which there was extreme motor excitement. Five were cases of agitated melancholia, and the rest were puerperal mania, periodic mania, or senile dementia, and all the patients were restless or noisy. There was no patient in the hospital suffering from general paresis, but I have no doubt the drug would be of value in the maniacal outbreaks of this disorder. In all cases it produced drowsiness and quietude, followed in a short time by sleep of a natural character, free from bad dreams and bad after-effects. The sleep lasted about six or seven hours, and the patients awoke less restless and agitated.

One patient of the most maniacal character well demon-

strated the value of the drug. The woman, aged 56 years, having been treated at another hospital for the insane for acute mania, was removed in an exalted but improved condition after about twelve months' treatment. After a residence of a few days at home she suffered a relapse, became extremely violent, and it was absolutely necessary to remove her to the hospital. When admitted she was very restless, refused to eat or keep her clothing on, and resisted everything done for her comfort or convenience. Her resistance was so great that it required six nurses to hold her while she was fed by the stomach tube. Bromidia, paraldehyde, sulfonal and trional were each used in succession to quiet her, but only produced sleep for a few hours. Under 40 grains of chloretone she became stupid and slept; on awakening was much quieter. The dose was repeated when necessary, and in a week she took her food voluntarily, was quiet, tidy and able to answer questions. Since that time she has suffered from another maniacal outbreak, but the length of the attack was reduced very appreciably by the use of chloretone.

In the cases of agitated melancholia the results obtained were all that could be desired. In one patient, transferred from another hospital, with a history of having been insane for eighteen months, suffering from depression, restless, tearing her clothing, in constant motion, wringing her hands and crying, refusing food and resisting all attention, the chloretone was effective after the first dose of 20 grains, and was repeated only at intervals of thirty-six hours thereafter, and she is beginning to show marked mental improvement. In this case also various other hypnotics were used without benefit. In the remaining cases the drug was equally effective, and although it was only necessary to use it a few times, yet it showed its value in quieting those patients with insanity in whom restlessness and agitation are prominent symptoms.

In summing up the results and effects obtained by the administration of chloretone in the limited number of cases in which it was employed, the following deductions can be made:

It is most valuable and serviceable in the motor excitement of acute maniacal attacks and of agitated melancholia.

It could very probably be used with benefit in the motor excitement of general paresis. The fact that it does not depress the heart action ensures for the drug special consideration in all cases of acute insanity with restlessness where it is necessary to preserve all the vital forces to tide the patient over the attack and prevent death from exhaustion. In this respect it is not so dangerous as bromidia or sulfonal, which are depressants. It is more agreeable to take than paraldehyde and does not leave bad after-effects.

Below are given clinical reports compiled from the histories found in our case-books:

Case 1. Male inebriate, drug habitué, not excited, sleepless, accustomed to 45 grains each of sod. brom. and chloral hydrate every evening. Given 25 grains chloretone; no effect.

Case 2. Male; senile dementia; restless, excitable, sleepless. Given 20 grains chloretone at bedtime on two successive nights. Patient slept well. Not given succeeding nights. Remained quiet.

Case 3. Male, colored, forty-five years, acute mania, insane seven months, noisy, excitable, talking in loud voice during the night. Fifteen grains chloretone given with little apparent effect; symptoms persisting. Increased dose to 30 grains once a day, producing sleep for eight to ten hours; refreshed (not depressed) on awakening. Is now taking no drug and remains quiet.

Case 4. Male, hypochondriac, melancholic, sleepless, had been given 40 grains sulfonal, which produced sleep for about four hours. Twenty grains chloretone gave sleep from 11 P. M. to 6 A. M. This repeated with equal success until spring, when, with daily labor out of doors, he needs no drugs to gain sufficient sleep. Has persistent delusion that stomach is occupied by snakes.

Case 5. Male, religious melancholia, with alternate stages of depression and exaltation; in exalted state was noisy, talkative, sleepless. Received several small (10-15 grain) doses chloretone with no apparent quieting effect. Dose increased to 30 grains gave six to eight hours' continuous sleep, awaking quiet. This continued for four or five evenings, patient giving no trouble to present time.

Case 6. Male, recurrent mania; been admitted to hospital four times. When last admitted was quiet for a few days; then became excited, preaching, shouting, very noisy and troublesome. Small doses (10-15 grains) had no apparent

effect; 35 grains given three or four nights in succession produced sleep each time all night until morning call, 6.30 A. M. Quiet after fourth night and finally sent home recovered.

Case 7. Male, recurrent mania, religious delusions; quiet on admission to hospital, in a few days grew noisy and troublesome, fighting and not sleeping at night. Twenty-five grains chloretone were given for two nights, producing sleep, after which could be removed to a quiet ward; steadily improved, and has now (two months later) gone home.

Case 8. Female, recurrent mania; attacks usually last about two weeks, during which time she is excited, quarrelsome, abusive and noisy, with insomnia. Twenty grains chloretone not giving sleep, but having a decided quieting effect lasting twelve to fifteen hours, with some sleep at night. This was repeated at intervals of two or three days until the attack had abated. This was an unusually troublesome case in which it was not desired to produce complete hypnosis during the day so long as the patient remained quiet during the day and slept at night. No other hypnotic was given.

Case 9. Female, aged forty-six, climacteric insanity, delusions of such a character as to cause at long intervals decided excitement with destructive tendencies. A small dose (15 grains) of chloretone given on first appearance of the outbreak produced sleep for several hours, and on awaking she was tractable and amiable.

Case 10. Age fifty-eight, melancholia agitata, delusions of persecution of a most painful nature. Twenty-grain doses chloretone gave decided relief for several days, with sleep of an apparently refreshing character at night.

Case 11. Female, acute mania, greatly excited with delusions and hallucinations, constantly tearing clothing, untidy in her habits, walking the room day and night, complete insomnia. One 30-grain dose of chloretone materially quieted her. This repeated on the second day, produced such complete and profound hypnosis for four days that a stomach tube was passed and the forearms were freely incised for the relief of an extensive and previously sensitive cellulitis, without any discomfort or resistance from the patient.

Case 12. Female, age thirty-four, primipara, puerperal melancholia, much agitated, constantly walking the floor, disrobing, pulling out her hair, moaning and crying. Bromidia and paraldehyde both given with almost no effect. It is probable that from continued use of these drugs for two years at intervals she had gained some insusceptibility to their action. First dose of chloretone given was 20 grains, which quieted

her for eight or ten hours. A repetition of the dose twenty-four hours after the first gave about sixteen hours' sleep, and she was much quieter and apparently refreshed when she woke up; remained quieted for twenty-four hours, when the dose had to be repeated; but gradually her symptoms have abated. It is quite certain that immediate sleep could have been induced in the first instance by a larger dose, but it was not deemed desirable.

Case 13. Female, age forty, subacute mania with great motor excitability, moving about incessantly day and night; tried both bromidia and paraldehyde with some good effect, but not so marked as was subsequently produced by 20-grain doses of chloretone. This patient now (six weeks later) is entirely free from the mentioned motor excitability and otherwise quiet.

Case 14. Female, age thirty-two, chronic mania, mostly motor excitement with destructive tendencies. All medicine and food had to be given through tube. Bromidia, paraldehyde and chloretone, all seemed equally ineffective. This is a chronic case of two years' duration.

Case 15. Female, age thirty-nine, chronic mania, constantly excited, destructive and violent. Thirty-grain doses chloretone produced profound hypnosis lasting from eight to twelve hours. Smaller doses have a very decided quieting effect.

Case 16. Female, age thirty-four, recurrent mania; attacks last about two weeks, during which time she is noisy, violent and destructive. Thirty-grain doses of chloretone produce the usual happy results, and are repeated at intervals of two days until the attacks subside.

Case 17. Female, age twenty-four, acute mania, great excitability, very noisy, quarrelsome, violent and destructive. On being given 30-grain doses of chloretone through stomach tube she becomes a model patient, quiet and tractable, and remains so for the next twelve or fifteen hours.

Case 18. Female, age thirty-six, melancholia, much depressed, greatly excited, delusions of persecution. After 30-grain doses of chloretone she becomes quiet and gets along nicely for the next twelve to fifteen hours.



## Society Reports.

### THE PHILADELPHIA NEUROLOGICAL SOCIETY.

March 26, 1900.

The President, Dr. William G. Spiller, in the chair.

#### AN ATYPICAL CASE OF ACROMEGALY.

Dr. Frederick A. Packard showed a middle-aged man who presented many of the features present in acromegaly. The patient had been an excessively heavy drinker, had lived a very irregular life, and had extremely unclean habits. In his family history there was nothing to indicate the presence of similar trouble in any of his antecedents. Careful questioning of his family produced no evidence of any difference in the appearance of his face or in the size of his extremities during the last few years. The patient had complained somewhat of headache, and was evidently of a low grade of intellect. There were no symptoms directly pointing to organic disease of the central nervous system. There was no choking of the disks and no alteration of the field of vision. The face was very heavy, with projecting eyebrows, prominent malar bones and some prognathism. The clavicles were very large and heavy, and there was some dorso-cervical kyphosis. The hands were distinctly spade-like in appearance, with marked prominence of the thenar and hypothenar eminences, while on grasping the hand the tissues gave the same doughy feeling as is present in more marked cases of acromegaly. Both legs were greatly swollen, apparently from prolonged inflammatory obstruction, doubtless produced by frequent infection through ulcers, of which many scars were visible. The case was shown as a probable example of acromegaly of a mild grade and not progressive. The absence of evidence of involvement of the pituitary gland was not considered sufficient to exclude the diagnosis of acromegaly, and was rather in keeping with the absence of progressive signs of structural change in the extremities.

Dr. F. X. Dercum said that while it was difficult to express a definite opinion, the case looked like one of acromegaly, probably not very far advanced. It was not a typical one.

Dr. D. J. McCarthy stated that his remembrance of this individual dated back ten or fifteen years, and that his appearance then was the same as it is now. He thought that the case was probably not one of developing acromegaly.

Dr. W. G. Spiller remarked that a case of acromegaly might reach a certain stage and not attain full development (*forme fruste*). Dr. Packard had not referred to drowsiness, which is present in many cases of acromegaly. In a case from Dr. Spiller's clinic, presented by

Dr. Witmer, the patient, a woman, would go to sleep while eating. The absence of bi-temporal hemianopsia did not torbid a diagnosis of acromegaly. In several cases that Dr. Spiller had seen this sign had been absent. This absence is noteworthy because in many cases the pituitary body has been found involved.

Dr. F. A. Packard said that what Dr. McCarthy stated bore out the testimony of the man's family. They admit no change in his appearance for many years. There had been no somnolence while he had been under observation, and there was no history of it.

In regard to hemianopsia, he said that in a patient he had reported some years ago there had been bi-temporal hemianopsia in 1885, and in 1892 there was only symmetrical contraction of the fields of vision. Both examinations had been made by Dr. de Schweinitz. The explanation of this fact was not clear to him, as the man had a huge pituitary body.

Dr. W. G. Spiller said that he had examined the brain and optic nerves from the case referred to by Dr. Packard. There was distinct atrophy in one optic nerve. He was inclined to attribute the hemianopsia, which had appeared and disappeared, chiefly to pressure, relieved later by erosion of bone at the base of the skull.

#### NOTE ON A CASE CLOSELY RESEMBLING THOMSEN'S DISEASE—PARAMYOTONUS.

Dr. F. X. Dercum presented a bricklayer, age 40 years, married. His family and previous history were negative and unimportant. Alcoholism and venereal disease were denied. About two years ago he began to have cramps in the calves of both legs, the posterior aspects of the thighs, and in the back between the shoulder blades. These cramps were always provoked by muscular effort. They have recurred with increasing frequency, and at present are so marked that whenever he attempts to lift anything, or to extend his body and arms in the act of reaching, a severe cramp comes on in the regions described, and lasts for about two minutes. Of late the cramp has become so severe as to be painful, indeed, so much so that he has been compelled to quit work. The knee-jerks are normal. Sensation is normal. There are no signs whatever of organic disease. An examination of the eyes is negative. General health is excellent. The muscular spasm makes its appearance especially after prolonged rest. It is particularly marked in the morning. The muscles affected by the contractions are hypertrophied. An electrical examination was not made.

#### A CASE OF LANDRY'S PARALYSIS WITH RECOVERY.

Dr. Wm. G. Spiller reported a case that had been referred to him by Dr. Allman. The patient, a young woman, had attempted to bring on a miscarriage and had a purulent uterine discharge as a result. She became paralyzed one evening in the lower limbs, and by the following morning was almost completely unable to move any of her extremities. Dr. Spill-

er had seen her about one week after the beginning of the paralysis. She could move the toes very slightly, and raise the legs only a few inches from the bed. She had a little power in the hands, but the upper limbs were almost completely paralyzed. Electrical reactions were normal, except that a somewhat stronger current than usual was required to produce contractions. The knee-jerks were lost. Sensation was not affected, and no disturbance of the vesical and rectal functions had occurred. The patient was fully conscious and very anxious about her condition. She did not have any spontaneous pain or pain on pressure. Within two or three weeks after the first symptoms of the disease had appeared improvement began, and the patient made a complete recovery from the paralysis.

Dr. F. S. Pearce said with regard to prognosis, that neuritis or poliomyelitis following infection from the uterus is likely to be very grave. Two cases that he had seen had ended fatally. He thought that Dr. Spiller was to be congratulated on the result in his case.

Dr. Stewart Paton, by invitation, read a paper on the hospitals for the insane and the study of mental diseases.\*

Dr. E. N. Brush, of the Sheppard and Enoch Pratt Hospital, gave some facts in regard to the institution. It was founded by a Quaker, who died in 1857, who was manifestly ahead of his time, and, among other things, in regard to the method of caring for the insane. His will gave his entire estate to the trustees untrammelled by any conditions. He, however, left some suggestions stating that he wanted a hospital built where the attempt should be made, regardless of expense, to advance the condition of the insane. The amount left was about \$600,000. The hospital was opened about eight years ago. Two and a half years ago the will of the late Enoch Pratt left the trustees of the Sheppard Asylum his residuary estate, if they would change the name of the institution to the Sheppard and Enoch Pratt Hospital. The trustees have been remarkably broad-minded men, and have sought the best advice they could obtain, and have planned not only to care for the wards of the institution, but to carry out the idea of the founder by advancing the care and treatment of the insane in all directions. Dr. Paton had voiced very clearly the ideas of those who have looked into this matter, viz., that it is not alone the laboratory, but that the laboratory is a minor matter and sometimes a drag upon the hospital, unless the hospital has combined laboratory and clinical workers who intelligently study the problems in the case. What the future shall develop, the future alone can tell, but it is the intention of the trustees to place before the profession the wards of the hospital as places where, under proper direction, scientific research in subjects connected with psychiatry and allied departments of medicine may be made.

Dr. F. X. Dercum said that while all appreciated the importance of detailed clinical studies, especially in the insane, yet in such cases the difficulty was very great, especially in institutions. In many cases it is impossible to do more than make a tentative diagnosis. It is

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\* This paper has been published in the Philadelphia Medical Journal, May 26 and June 2, 1900.

often impossible to obtain the history from the relatives or friends, and the study of the symptoms other than mental is extremely difficult, on account of the mental condition of the patient. He thought that the importance of the clinical laboratory was greater than that of the pathological laboratory. Psychiatrists should study their cases from the standpoint of neurology and internal medicine. A complete visceral study should be made, just as there should be a complete study of the nervous and mental manifestations. It not infrequently happens that the mental affection is the result of some internal or general disease, and not a primary disease of the nervous system.

The President said that the necessity of increased study of mental disease is apparent, and all had listened with great pleasure to the remarks of Dr. Paton bearing upon that point. The union of clinical and pathological study is very important. In parietic dementia, which possibly has been better studied pathologically than any other mental disease, the pathology is not fully known, but there are some who from the pathological condition alone are willing to make a diagnosis. He was not inclined to believe that all the causes of mental diseases could be revealed by the microscope. There is probably much more in mental diseases than can be discovered by a study of the pathological conditions alone. The brain is very susceptible to various substances and probably certain alterations cannot be detected by the microscope. Physiological chemistry may be of service in the study of mental affections, and probably some of the mental symptoms produced by the infectious fevers are the result of chemical changes.

- 90 ACROMEGALIE ET DEGENERESCENCE MENTALE (Acromegaly and Mental Degeneracy). F. Farnier (Nouvelle Incon. de la Salpêtrière, Sept., Oct., '99).

This article is the result of the study of hereditary antecedents, psychical symptoms, the neuroses and the stigmata of degeneracy found in cases of acromegaly. The case quoted is a typical case of this disease with the curious complication of epilepsy and a condition approaching dementia. The author does not believe that this case can be explained by mere coincidence, but that degenerative states are very favorable for the development of acromegaly. A view of all cases in literature from this point of view is noted under the heads of heredity, diseased condition of character and intelligence, psychoses and neuroses. A résumé of the facts derived from this study are as follows: Nervous and mental diseases are frequently found in the families of individuals suffering from acromegaly. The patients themselves often present diverse manifestations of degeneracy all the way from intellectual enfeeblement to complete dementia; from melancholic depression to the systematized ideas of persecution, and from psychical stigmata to microcephalic skull with idiocy. Hysteria and epilepsy are found associated with acromegaly, as is also diabetes. This conclusion is finally drawn by the author: The neuro-arthritis or vesanic heredity offers a favorable soil for the development of acromegaly. This in its turn by certain disturbances which it produces in the secretion of ductless glands, as the hypophysis, reacts upon the nervous system, already in a state of instability by the hereditary influences noted above. This determines, according to the individual predisposition, various mental or nervous affections, always, however, in relation to the state of degeneracy. A very complete bibliography is affixed to the article.

SCHWAB.

## CHICAGO NEUROLOGICAL SOCIETY.

March 7, 1900.

The Vice-President, Dr. Hugh T. Patrick, in the chair.

The Society held a joint session with the Chicago Medical Society, for the discussion of brain tumors.

### THE GENERAL SYMPTOMS OF BRAIN TUMOR AND THE DIFFERENTIAL DIAGNOSIS.

Dr. Archibald Church read a paper with this title. He said that the principal symptoms of a growth within the skull are: Headache, generalized and localized, Jacksonian convulsions, mental impairment, choked disc, optic papillitis, vomiting and vertigo. According to Knapp, in 401 out of 614 cases headache was present. Twenty-three cases of tumor of the brain, verified by operation or by post-mortem examination, or both, have been observed by Dr. Church, and twenty out of these twenty-three cases presented headache in a marked degree. The character of the headache which we find in brain tumor assists us in making the diagnosis in many instances. In some cases the headache is rare, or when present is trifling; in others the headache is very intense. The headache of brain tumor is generalized; it is not strictly confined to the temporal, the occipital, or lateral regions, though in some cases it may be, and it is very difficult in children to get a distinct description of its character and location. In certain cases, where the headache is circumscribed and characterized by tenderness of the skull on pressure, it serves as a localizing feature. In one case a frontal headache was associated with tumor in the posterior portion of the temporo-sphenoidal lobe. In another case of frontal headache the tumor was found in the cerebellum, and in other instances where the headache has been occipital, the tumor has been found in the frontal regions. Ordinarily the patient first complains of headache. It is a generalized symptom, and may be a slight variation from normal health. The diagnosis of headache entails one of the most difficult problems the physician encounters. Some headaches which have been confounded with those of brain tumor are migrainous. Migraine is a neurosis which, of all the neuroses, shows, as a rule, a marked heredity. Patients will tell us that their father, mother, or grandparents had migraine. A history of migraine, coupled with the clinical features of the case, should be sufficient in some instances to make the diagnosis. A migrainous patient may have brain tumor. Other patients with severe headaches are those who suffer from neurasthenia. Patients with neurasthenia or

hysteria may have brain tumor; and it is only after making a careful examination and eliciting the history of the case that one may be justified in ascribing severe headaches to hysteria or to neurasthenia alone.

The second major symptom of brain tumor is choked disc. As a rule, it is a symptom which is not of early appearance in brain tumor, consequently it is not a sign in the early stages of the disease. Indeed, the presence of choked disc either on one side or the other, or on both sides, is presumptive evidence of the presence of brain tumor, and when coupled with even two or three of the other major symptoms it may serve to confirm the diagnosis of a tumor within the skull. It is found at one time or another in over 80 per cent. of the cases.

A defective mental state is seen in many cases of brain tumor, and usually develops rather late in the course of the malady. In Dr. Church's twenty-three cases, seventeen showed the symptom late. Almost invariably the patient is apathetic. Sooner or later he is more or less indifferent to surroundings and conditions. He manifests mental sluggishness; he does not apprehend promptly the questions that are put to him; he takes time to think and to express his thoughts, and during this prolonged period of slow mental action he loses the drift of the question altogether, and may require a repetition of the question in order to respond in a proper manner. In certain cases the stupor is pronounced. The patient becomes lethargic, sometimes comatose.

*A priori*, it would seem that tumors located in the prefrontal region produce more disturbance of the mental processes than those located further back. This may or may not be true.

Fully one-half of the cases show convulsions early in the disease. In some instances the convulsions are preceded by intense headache; in others by vomiting. In the majority of cases they are followed by vomiting, and the larger proportion of cases which show general convulsions also manifest slighter attacks, such as Jacksonian fits, momentary absence, sudden explosions in the ear, etc.

Vomiting is a common general symptom of brain tumor. Jacobi reports it to have been present in 172 out of 568 cases. It was present in 13 of Dr. Church's 23 cases. Ordinarily, where vomiting is due to brain tumor, it is likely to be of the projectile variety. This vomiting may occur irrespective of a clean tongue, indigestion, etc. It may be protracted, with decided nausea. Vomiting is most common in tumors of the brain that are located posteriorly, *i. e.*, those that involve the

peduncles of the cerebellum; those affecting the ocular apparatus, or involving the optic radiation, or those affecting the auditory apparatus of the internal ear or the nerves passing to the internal ear. In cases of brain tumor, with aural vertigo, vomiting is not uncommon. In the majority of cases the vomiting is persistent, and intracranial pressure becomes high. In other instances it occurs as an exception very early in the history of the case.

Vertigo occurred in 31 per cent. of the cases collected by Mills and Lloyd. In the twenty-three cases observed by Dr. Church vertigo was marked in seventeen. It is an early symptom and likely to be present when the ocular or aural apparatus is involved. It is associated with headache. Severe headache is followed by vertigo, or contrariwise; or vertigo is followed by vomiting, or *vice versa*. Muscular efforts, depending upon the position of the head, a sudden turning in bed, or other disturbance, may produce vertigo and lead to vomiting.

In addition to the symptoms described, there is a condition of the head which is not sufficiently dwelt upon, and which in some cases serves to make the diagnosis conclusive, and that is a certain degree of hydrocephalus, particularly in those who have not arrived at the age of puberty, and in whom the sutures are easily separated.

In some cases the tumor is so situated as to cut off the return circulation from the face and scalp; in other cases, where aneurysm exists, there is increased arterial tension.

Dr. Church makes it a routine practice to auscult the head of every patient who presents symptoms of a cerebral character, because in time something may be gained by it in cases of brain tumor. Auscultation in conjunction with percussion can be very easily done. By placing the stethoscope over the patient's brow one is enabled to detect changes in the percussion note which may in some instances be of undoubted value.

In a few cases tumor of the brain has produced erosions of the skull. Mention was made of a case seen with Drs. Fütterer and Henrotin, in which a tumor in the prefrontal region had eroded the temporal bone to such an extent that the end of a finger pushed through the skull on one side, after the death of the patient, could be easily detected by palpation on the opposite side. Westphal has reported a number of cases of erosion of the skull from brain tumor, followed by the spontaneous escape of fluid either into the temporal fossa, the nose, or the antra of the face. It is well, therefore, to examine

carefully with the point of the finger every portion of the skull, and get as near as we can to the surface of the brain.

Percussion is another means of investigating the condition of the skull. Macewen, of Scotland, and Dana, of this country, believe they have noticed a distinct change in the percussion note over the seat of a cerebral tumor or abscess. A distinct dullness is elicited in these cases. Oppenheim and Bruns attach great importance to this method of investigation, claiming that if a circumscribed dullness can be elicited on percussion, it is a strong indication that a tumor lies in that area.

Mention was made of the value of the disappearance of the cracked pot sound in children, when the intra-cranial pressure was increased.

#### CEREBRAL LOCALIZATION.

Dr. Sydney Kuh gave a brief review of what is to-day known in regard to cerebral localization. The differential diagnosis between so-called idiopathic and Jacksonian epilepsy, and the general characteristics of cerebral palsies, were discussed in detail. The uncertainty of brain localization was emphasized particularly, and this point was illustrated by a number of interesting cases, partly taken from medical literature and partly from Dr. Kuh's own experience. Thus two cases were mentioned in which spasms occurred on the side of the body on which the cerebral lesion was found at the necropsy; one in which an angiosarcoma had destroyed practically all of the vermis superior cerebelli without causing any topical symptoms; one in which a psammoma of the hypophysis in place of causing acromegaly, was associated with stunted growth, and finally a case of cerebellar abscess with no cerebellar symptoms, but with typical Jacksonian epilepsy on the side on which the abscess was found. Dr. Kuh gave a brief history of one of those very rare cases in which laryngeal spasms were caused by cortical disease. His patient presented, besides the laryngeal spasms, spasms in the muscles of the face and a purely motor aphasia, and later right hemiplegia. Examination of the fundus showed atrophy of both optic nerves.

#### THE VALUE OF EYE AND EAR SYMPTOMS IN BRAIN TUMOR.

Dr. William H. Wilder placed optic neuritis second in importance to headache in the list of general symptoms of brain tumor, and yet, he said, it is surprising to note how often in the reports of such cases no mention is made of the condition of the optic nerve. Optic neuritis should be looked for at



once in all cases of suspected focal disease of the brain, for it may be present although normal central vision exists.

As to the frequency of this sign, the writer found mention of it in 104 cases out of 140 cases of brain tumor recorded in the literature for four consecutive years, this being about 75 per cent. It is safe to say that optic neuritis occurs in 80 per cent. of all cases of brain tumor. The records show that it is of somewhat more frequent occurrence in cerebellar tumors, being noted in 90 per cent. of such cases.

Although so valuable as a general sign, it possesses very little worth as a localizing sign, nor does it furnish any definite information as to the character or size of the growth. It may be present with small tumors, and may be absent with large ones. Inasmuch as the optic neuritis in some cases may be of a low grade, it is important for the observer to avoid mistaking for organic change the blurring of the outlines of the disc in the high degrees of astigmatism.

Certain general conditions causing optic neuritis, such as meningitis, anemia, chronic nephritis and plumbism should be carefully excluded before placing too much reliance upon this sign. Emphasis should be placed upon the importance of repeated examinations of the fundus of the eye during the course of every case of suspected brain tumor.

Another important general eye symptom of brain tumor, although less frequent than the former, is temporary periodic blindness, coming on in the early stages of the growth. The attacks of blindness are sudden, and last from a few seconds to half a minute or longer. In the intervals the vision is as good as before, but frequent attacks may lead to permanent impairment of sight, as was seen by Dr. Wilder in a case recently.

Hemianopsia may be an important localizing eye symptom of brain tumor. It may be absolute or relative, and coexisting with certain other symptoms such as mind-blindness, amnesic color blindness, word-blindness, etc., would point strongly to lesion of the occipital lobe. It must be differentiated from the hemianopsia occurring with lesions of the optic tracts. Wernicke's pupillary sign may aid in this.

The ear symptoms in brain tumor relate principally to the labyrinth and the auditory nerve, and consist of subjective sounds and varying degrees of deafness. Careful exclusion of middle ear disease is necessary before formulating any conclusion as to the relation of ear symptoms to a possible intracranial growth.

RECENT ADDITIONS TO THE TECHNIQUE OF OPENING THE CRANIUM FOR DIAGNOSTICATING AND REMOVING TUMORS.

Dr. Van Hook described the Wagner method of temporary resection of parts of the cranium, and the Doyen method of so-called temporary craniectomy. The former has for its object the raising of a flap of skin, muscle, pericranium and bone from the side of the skull, with a nutrient pedicle of the soft parts. The Doyen method consists simply in raising a very large flap of the same kind. After exploration, or the removal of a tumor, the flap is turned back into place and sutured.

The Creyer, Doyen and Van Arsdale saws, driven by electrical engines were mentioned.

Two modern devices for opening the skull were considered at once commendable and novel. The Gigli saw, consisting of a simple wire of steel twisted while hot to produce a roughened surface, is used to open the skull by making cuts about the plate of bone to be lifted. The wire saw is introduced through drill-holes. Dr. Van Hook objected to the saw because it acts inside the skull in an approximately straight line in the direction in which the skull is to be opened, like the chord of an arc, and the dura may be wounded unless great care is used.

On the whole Dr. Van Hook favored most the DeVilbiss-Dahlgren cutting forceps for making channels in the cranium. This instrument has the advantages of availability as to price, ease of transportation and sterilization, and makes a cut through the bone narrow enough to facilitate rapid healing without injuring the brain or its coverings.

Owing to the exhaustive character of the papers read, the discussion was very limited. Dr. Frank Billings related the history of a case in which operation failed to reveal a tumor, but the post-mortem examination showed a subcortical glioma under the motor center for the upper extremity, where the symptoms had already indicated its presence. Dr. Ochsner asserted that operative procedures in brain tumor were failures.

## Periscope.

### CLINICAL NEUROLOGY.

- 91 "EIN BEITRAG ZUR KENNTNISS DER PSEUDOLOGIA PHANTASTICA" (An Example of Pseudologia Phantastica). Redlich (Allg. Zeitschrift für Psychiatrie, 1900, LVII, 1, S. 65).

By this somewhat phantastic name, it has been proposed to designate "pathological lying," whose special characteristics the author gives as follows: It occurs always upon a degenerative basis, in imbeciles, hysterical subjects, etc. It comes on as an irresistible impulse, like an obsession as it were, and is often connected with a state of double consciousness, so that the patient, when in his comparatively normal condition, may have a realization of the misrepresentations which he has made, and may be much distressed thereby. There is generally a great susceptibility to auto-suggestion. The author discusses the subject at some length and gives the history of a typical case. ALLEN.

- 92 ACROMEGALY AND RAYNAUD'S DISEASE.

Of more than usual interest is A. Boettiger's article (*Munch. med. Woch.*, Dec. 19, 1899), giving an account of a case in which both these rare maladies were present. The patient (21 years of age), noticed as first symptoms, coldness, numbness and formication in the hands and feet which soon turned a dark-blue color. Neuralgic pains were absent. The paresthesiæ and discoloration, at first paroxysmal, soon became more intense and persistent. The patient's attention was soon attracted to an increase in size of the hands and head. Symptoms of intracranial pressure, eye-disturbances or rheumatic pains were absent, nor could any retinal or other ocular changes be demonstrated. The tongue was wider, the nose thicker, and a radiograph showed a decided increase in volume of the bones, and more particularly of the soft parts of the right arm and left hand. The skin of the palm was tough, hypertrophied and showed excessive cornification; the hands themselves were cold and moist, but the most prominent symptom was the bluish-black discoloration, mottled with a few irregular reddish spots, and extending above the wrists. The sensation of touch was perfectly normal, and pain was elicited only on penetrating for some distance into the skin. The susceptibility to heat and cold was much diminished.

In discussing the case the author says that it is not the first one of its kind; indeed, there often are peripheral vasomotor neuroses in the beginning stages of acromegaly. There are, in short, close relations between the latter disorder and Raynaud's disease, since stasis is not rare in the one and swelling and thickening may occur in the other. In both, the seat (hands, feet, nose, ears, and, later, arms and legs) is the same, and similar pathologic vessel changes have been discovered. Boettiger does not believe in the hypophysis theory of acromegaly, but on the basis of the cited case develops a new explanation. It seems to him illogical to refer so general a disease to so small an organ; then, too, the fact that the pituitary body is so often diseased without

acromegaly being present, and that symptoms directly referable to this body may be wanting in gigantism, make him conclude that the general belief is untenable. There are many arguments which favor the view that acromegaly is not caused by pituitary tumor. While there may be a certain relationship between the rapidity of growth of the latter and the amount of thickening in the bones, this is by no means constant. Granted that this disorder does not depend on a specific internal secretion, a cause for the hypophyseal hypertrophy must be sought. It is a well-known fact that poisons in the circulation, such as arsenic, alcohol, lead, etc., can cause changes in the peripheral parts of the body. There are also diseases dependent upon the production within the body in some unknown way of toxin which can lead to vasomotor and trophic disturbances in the same sites. Raynaud's disease has been considered as such. It is probable that, due to heredity, constitution, diet, etc., toxins may be produced which have a selective action on certain tissues. Thus arthritis deformans and hypertrophic pulmonary arthropathy involve the bones; chemical poisons, diphtheria, etc., the nerves; scleroderma and myxedema, the skin, etc. Acromegaly also begins in the periphery, and at its outset there may be symptoms of Raynaud's disease; it thus seems likely that leucomains are the essential cause. The analogy is further borne out by the cerebral symptoms which suggest a toxemia. But where do these toxins come from? Most, no doubt, would say the hypophysis, but for this Boettiger thinks there is no proof.

The case described was put on vegetable diet and treated with iodine, hydrotherapy, peripheral faradization and central galvanization with the most encouraging results.

JELLIFFE.

- 93 "UEBER DIE BEZIEHUNGEN DER AKROMEGALIE ZUM DIABETES MELLITUS" (On the Relation of Acromegaly to Diabetes Mellitus). Schlesinger. (Weiner klinische Rundschau, 1900, No. 15).

According to the author, diabetes may occur in acromegalic patients in two forms, the one differing little from diabetes in other persons, pursuing a regular course and unless modified by diet, and treatment, the quantity of sugar excreted remaining quite constant. In the other form, the quantity of urine and the percentage of sugar may undergo sudden variations independent of treatment or diet, the sugar sometimes disappearing entirely for a time, or being present only when large quantities of starches or sugar have been ingested. The first class of cases he would make dependent upon increase of connective tissue in the pancreas, in connection with the same process elsewhere. For the second, he would make the growth of the hypophysis, and the thereby increased cerebral pressure, responsible. Three cases whose clinical histories he gives would seem to support these views, but there are no autopsy findings given, as the patients are still living.

ALLEN.

- 94 "A CASE OF CEREBRAL ABSCESS WITH OPERATION." Leutert (Vereins-Beilage, No. 20 der Deutschen med. Wochenschrift, No. 21, 1900). In the Verein für wissenschaftliche Heilkunde in Königsberg i. Pr.

Leutert presented a patient who had had pain in the back of the neck, and right-sided headache. The pupils reacted promptly, and no paresis, no disturbance of sensation and no ankle clonus were observed. He had chronic purulent otitis media. The area about the ear was normal in appearance and not painful on pressure. Temperature was 36.1-36.2. No optic neuritis was detected, but there was some impairment of the mental functions. A history of nausea, vomit-

ing and constipation, lasting several months, was obtained. On the second day after the first examination, coma with loss of the corneal reflex developed, and then left-sided paresis was detected. At least 200 c.cm. of pus were obtained from the right temporal lobe by trephining. After six weeks the patient was discharged much improved.

SPILLER

- 95 "DIE CORRELATIVE EMPFINDLICHKEITS-SCHWANKUNG" (The Correlative Variation of Sensibility). Strausky and Ten Cate. (Wiener klin. Rundschau, 1900, XIV, 15, S. 290).

The authors put to themselves the following questions: 1. Does the production of *hypoesthesia* in one skin area produce *hyperesthesia* in another region?

2. If it does, is it in a symmetrically situated area and in what relation do the respective areas stand to each other?

3. Is this relationship such that homology with the so-called "transfer" is conceivable?

4. What are the conditions in pathological cases?

The results obtained in the examination of 40 cases, half healthy, half having nervous diseases, are utilized. Local anesthesia was produced by ethyl chloride, by Scheich's solution, and by ice. The hair esthesiometer of von Frey was used. Their conclusions are as follows:

1. In healthy persons without sensory disturbance, production of local hypoesthesia gives rise to *hyperesthesia* for touch and pain, in other skin areas, both on the same and on the opposite side. This they designate "correlative variation of sensibility," and speak of its "projection."

2. This variation is most marked in those areas, whether on the same or on the opposite side, which are supplied from the same *spinal* segment.

3. In skin regions supplied from neighboring segments, some variation may occur, but it is much less marked, and in areas supplied from distant segments there is little or no variation even though they may be located nearly symmetrically with regard to the hypoesthetic area.

4. With the manifestations of "transfer" correlative variation of sensibility has nothing to do. The former seems to be of cortical origin.

5. On account of insufficient number of observations and lack of autopsies, the authors do not feel justified in drawing positive conclusions with regard to cases having already some sensory defect, but simply state what they found.

a. In 4 cases of segmental involvement of the cord, "projection" either *from* or *to* the skin supplied from the diseased segment, was impossible. Where the lesion involved the whole cross section, the areas supplied from the segments below this showed no projection. Where only the gray matter was involved the segmental areas, both above and below, reacted normally.

b. In 6 cases of neuritis, projection was impossible *from* or *to* the skin supplied by the affected nerve.

c. In 3 cases of functional hemihypoesthesia projection *to* the affected area was impossible, but was observed *from* it.

d. In 1 case of cerebral syphilis (meningitis) projection occurred as in a normal person.

e. In 2 cases of Basedow's disease no projection to the face, but in other parts of the body it occurred normally.

ALLEN.

- 96 DOUBLE SYNDROME DE WEBER SUIVI D'AUTOPSIE (Double Syndrome of Weber, with Autopsy). A. Souques (Nouvelle Iconographie de la Salpêtrière, 13, 1900, p. 173).

The syndrome of Weber presents itself usually in a simple form, as the result of a unilateral lesion of the peduncular bulbar region. Following a sudden apoplectiform seizure, the patient was brought to the hospital with a left hemiplegia and a paralysis of the third nerve on the right side. This ocular paralysis was complete, with ptosis, external strabismus, immobility of the eye-ball, dilated pupils, no reaction to light nor to accommodation. In addition there was a right-sided hemiparesis and left ocular symptoms. Paresis on the left side was more marked than on the right; reflexes were normal. The urine contained albumin, and the patient was more or less stuporous all the time, dying finally of marasmus. The autopsy showed the presence of a bilateral lesion in the peduncular region. In the right cerebral peduncle were two foci of softening, separated by the locus niger, which appeared intact; one was situated in the red nucleus, involving the ocular-motor fibers, the other in the internal quarter of the foot of the peduncle. In the left peduncle a sclerotic focus of small dimension was found, involving the internal quarter of the foot of the peduncle. The nuclei of the oculo-motor were not affected, though the cells of the right nucleus seemed smaller than those of the left. The fibers of the right ocular-motor were totally degenerated; those of the left nerve were nearly normal. Examination of the arteries elsewhere showed evidence of an arteritis obliterans. The explanation of the clinical symptoms is clear from all these findings. The origin of the arteritis was not discovered. SCHWAB.

- 97 ANGIOME SEGMENTAIRE (Segmentary Angioma). G. Gasne and G. Guillaïn (Nouvelle Iconographie de la Salpêtrière, 13, 1900, p. 169).

A case which presents the following peculiarities: A woman sixty-three years old, who since birth has had a vascular ectasis of the right upper extremity and of the lateral thoracic region of this side. This ectasis shows itself by a large number of tumefactions of various sizes; some are reducible, others not; some are very soft, others give the sensation of a stringy mass. These tumors are spontaneously painful and are slightly sensitive upon pressure. They show no fluctuation, no souffle, no fremitus, and no vibrations. The other organs are normal. There exists then a general vascular sclerosis, which is congenital. The following conditions are important from the standpoint of different diagnosis: Arterio-venous aneurism, cirroid aneurism, serpentine arterial dilation, lymph-angioma, cutaneous angioma; these are all excluded and the authors believe that this case is one of congenital subcutaneous angioma. The pathogenesis of this condition is absolutely unknown. That it is possibly of nervous origin the segmentary distribution would seem to indicate. A radiograph included in the article brings out clearly the subcutaneous origin and the presence of concretions. SCHWAB.

## Book Reviews.

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DIE ERKRANKUNGEN DES NERVENSYSTEMS NACH UNFÄLLEN, MIT BESONDERER BERÜCKSICHTIGUNG DER UNTERSUCHUNG UND BEGUTACHTUNG. Von Privatdocent Dr. Heinrich Sachs und Dr. C. S. Freund, Nervenärzte in Breslau. Mit 20 Abbildungen im Text. Berlin W., 1899. Pp. 581.

It has been said with justice that it is more difficult to write an acceptable short book than an equally acceptable long one. The authors of the volume before us have made no effort at condensation, and the result is a somewhat tedious treatise of over 550 pages on a well-worn subject. One's feeling grows stronger with each page read that if there were fewer words and less unnecessary detail regarding matters known to every intelligent physician, the book would gain materially in force and usefulness. The hope is expressed in the preface that the book may be of use to the general practitioner of medicine, who may be called upon to give expert testimony in accident cases, as well as to the more accomplished neurologist. To this end many matters are introduced in the text which would not otherwise find a place, as, for example, details relating to the anatomy and physiology of the nervous system, and methods of examination. This aim, so often attempted, of reaching two classes of readers widely separated in their medical training, is not, in this volume, satisfactorily attained. The specialist, in his search for fundamental principles, is annoyed by a mass of perfectly commonplace and to him self-evident details, whereas the so-called general practitioner would certainly not go to so ponderous a book for aid in the formation of his opinion. It is to be regretted, therefore, that the book was not written with the avowed purpose of appealing to experts; in that case, we should no doubt have had a briefer and more readable treatise.

The authors write on the basis of a large experience and matured judgment in dealing with this always difficult class of legal cases; in all, they have made observations in approximately 30,000 cases following accident, certainly a sufficient number upon which to base conclusions of moment. As we have already indicated, not only is the general subject covered, but numerous collateral matters are introduced, which extend the scope of the book to a considerable degree. The views of the authors are in accord with the weight of present opinion regarding the nature of the neuroses following accident. They take the conservative ground regarding simulation which must now be regarded as established, and which they express as follows: "Eine absolut reine Simulation bei ganz gesunden Menschen ist etwas äusserst Seltenes" (an absolutely pure simulation in perfectly normal persons is exceedingly rare).

Regarding terminology, the phrase, "traumatic neurosis," is conspicuous by its absence. The authors believe it a dangerous because a convenient term, and are urgent that when we are ignorant of the exact diagnosis in a given case we should use a circumlocution rather than employ the easy method of terming it a "traumatic neurosis." This explanation is made with a certain apology to Oppenheim and a recognition of his services in the investigation of the various morbid conditions included under that name. Sixty-nine cases are cited in considerable detail, illustrative of the various predominant forms in which the accident neuroses may manifest themselves. This is certainly a practical and valuable portion of the book. Throughout the text there

is a complete avoidance of references, either by means of foot notes, or by numbered index references. There is, however, an index of the literature appended independently, which fills about ten pages of small type. In spite of this seeming completeness one is immediately struck with the almost entire neglect of the English and American literature. In fact, so far as we are able to see, no reference whatever is made to any work on the neuroses following accident, excepting to a German translation of Page. In consideration of the evident character of the book as a complete exposition of the subject under consideration, and in view of the fact that so portentous an array of references has been collected, we find that such an omission is not to be lightly excused.

E. W. TAYLOR.

#### BOOKS RECEIVED.

DIE CASTRATION IN RECHTLICHER, SOCIALER UND VITALER HINSICHT BETRACHTET VON DR. CONRAD RIEGER. Jena: Gustav Fisher.

ELEMENTS OF CLINICAL BACTERIOLOGY. By Ernst Levy and Felix Klemperer, M.D. Philadelphia: W. B. Saunders.

ESSENTIALS OF MEDICAL DIAGNOSIS. By Solomon Solis-Cohen and Augustus A. Eshner, M.D. Philadelphia: W. B. Saunders.

THE MEDICAL DISEASES OF CHILDHOOD. By Nathan Oppenheim, M.D. New York: The Macmillan Co.

A HANDBOOK FOR NURSES. By J. K. Watson, M. D. Philadelphia: W. B. Saunders.

98 LA POLYNÉVRITESYPHILITIQUE (Syphilitic Polyneuritis. R. Cestan (Nouvelle Iconographie de la Salpêtrière, 13, 1900, p. 153).

A study of two cases of syphilitic polyneuritis and a consideration of eleven other cases found in literature form the basis of this article by R. Cestan. Some of the cases found in literature are open to the criticism that, on account of the prolonged use of mercury, the polyneuritis may be due to that agent and not to the syphilis. Those cases only should be regarded as purely syphilitic in origin in which the polyneuritis has appeared shortly after the initial lesion or before mercury has been given in great quantities. In this way the number of cases of undoubted syphilitic polyneuritis are very few. The disease appears in three forms, motor, sensori-motor, and pseudo-tabetic. All the cases hitherto reported, including those of the author, have a favorable prognosis. The condition never runs a rapid ascending course. It develops sometimes as a slowing ascending form or as a disseminated form, never being accompanied by psychical symptoms. The facial nerves do not seem easily affected by the process. As in other forms of polyneuritis, the differential diagnosis must be made from central lesions of the cord due to syphilis, acute myelitis, poliomyelitis, etc. The diagnosis is made by exclusion and by the favorable effect upon the neuritis of antisiphilitic treatment.

SCHWAB.



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THE  
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**Original Articles.**

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DISCUSSION ON THE NEURONE DOCTRINE IN ITS RELATIONSHIP TO DISEASE OF THE NERVOUS SYSTEM.

(a) THE ANATOMIC-CYTOLOGICAL RELATIONSHIP OF THE NEURONE TO DISEASE OF THE NERVOUS SYSTEM.\*

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In view of the papers which are to follow, the present one may perhaps be most serviceable if it be limited to a statement of some of the principal facts of the general anatomy of the nervous system as at present understood, and a few remarks with regard to the nature and significance of the neurone doctrine in connection with these.

It may be considered as established to-day that the nervous system, like all other parts of the body, consists of cells and of intercellular substances. Aside from the covering membranes of the brain and spinal cord, the blood and lymph vessels of the nervous system, and the slight amount of connective tissue which accompanies them, as well as the cellular sheaths of the axis-cylinder processes and cell-bodies of the peripheral cerebro-spinal nerves and of the sympathetic system, all of the morphological elements in the nervous system are to be regarded as derivatives of the ectoderm of the embryo. Among

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\*Read at the twenty-sixth annual meeting of the American Neurological Association, May 1, 2 and 3, 1900.

these ectodermal derivatives can be distinguished two principal varieties of cells:

- (1) That which includes the supporting elements of the nervous system, namely, the neuroglia and the ependyma, and
- (2) that which includes the true nerve-cells or neurones.

The supporting cells and the true nerve cells are very simple in form in the beginning of development; later on, they undergo remarkable changes in form, distribution and arrangement. The ependyma cells are transformed to the long-drawn out structures so familiar in Golgi preparations. The neuroglia cells assume variable shapes, and give rise to specific cell-products—the so-called neuroglia fibrils—parts of which may even become extra-cellular. The true nerve-cells or neurones wander for shorter or longer distances from their breeding-places; they are sometimes aggregated in groups or layers, sometimes very irregularly distributed; they exhibit extraordinary variations in form, the protoplasm becoming manifoldly divided so as to give rise to (1) a relatively abundant mass—the so-called perikaryon—containing the nucleus, centrosome and archiplasm, (2) a group of relatively short processes—the so-called dendrites—resembling the perikaryon closely in minute structure, and (3) one or more processes which do not divide until at some distance from the cell-body—so-called axones—which differ somewhat in external form and internal structure from the dendrites, in that they are in general more delicate, are devoid of the so-called tigroid substance, and often extend for great distances from the perikaryon, in which event, except in the sympathetic nervous system, they become surrounded for the larger part of their course with a fatty or myelin sheath.

Inside a single tissue there is perhaps nowhere in the body as much variability in the elements in different parts as in nerve tissue. Muscle-cells are strikingly alike in all the voluntary muscles of the body, liver-cells in the right lobe are indistinguishable from those in the left lobe. Even in the kidney, where there are distinct variations in the cells lining different parts of the tubules, the general habitus is very similar.

The nerve cells of various parts have, however, a specific stamp; they can be recognized at a glance; they subserve speci-

fic functions. Like other cells in the body, except those of the blood and perhaps a few others, each nerve-cell or neurone, though possessing an individuality of its own, stands in intimate anatomical and physiological relations to other cells. The cells of epithelial tissues, for example, are related to one another by direct contact or by means of intercellular protoplasmic bridges, or by both methods. Many cartilage cells are connected with one another by fine protoplasmic threads. The nerve-cells or neurones are occasionally connected with one another by coarse strands of protoplasm (especially in the retina), much more often by means of intimate protoplasmic relations existing between the terminals and collaterals of the axone of one nerve cell or neurone, and the dendrites and perikaryons of other nerve-cells or neurones. Several forms of the latter relation have been studied: (1) the large calyx-like expansion of an axone terminal upon a cell-body (perikaryon) with apparent fusion or concrescence of the protoplasm of the two; (2) the intimate apposition and possible fusion of fine terminals of axones and collaterals with dendrites and perikaryons, sometimes apparently by means of a fine-meshed anastomosing network formed by the former, and (3) the passage of extremely minute, though uninterrupted, fibrils from one nerve-cell or neurone into another, or even through a series of nerve-cells or neurones, possibly through all the cell-units of the whole nervous system. Whatever may be the conclusion ultimately arrived at with regard to the interneuronal relations of the whole nervous system, one thing is certain, the relations between certain groups of neurones is very direct, so that we can distinguish firmly established conduction paths composed of superimposed and subimposed sets of nerve-cells or neurones. This is an organic memberment easily determinable by the methods at present at our disposal; its recognition has led to important advances in our knowledge of neural anatomy, physiology and pathology. The various afferent conduction paths, the motor conduction path from the cortex of the cerebrum to the voluntary muscles, the conduction paths upon the integrity of which many of the reflexes depend, as well as some of the more complex associative conduction paths are now tolerably well understood; the chains of nerve-cell

units or neurones, of which they are composed, have been localized; the integral links (or individual neurone systems) of each chain are being carefully studied.

A study of the historical development of a subject alone can give the clue for proper valuation of single contributions made to it. The influence of the introduction of the neurone-conception upon the shaping of neurological ideas as they exist at present can only be appreciated when the origin, course, and present status of that concept are considered. The history of the inception does not date back for a very long period, but it originated earlier than is generally supposed, having as it did a strong foundation in the minds of pathologists and embryologists many years before Ramón y Cajal began to publish the results of his work with the chrome-silver method of Golgi, and longer still before Waldeyer brought all the evidence in favor of the conception together, and gave to it a distinctive name.

The anatomical darkness in which investigators groped from the time of Soemmering (1798), to that of Deiters (1865), has recently been fully discussed by Stieda.<sup>1</sup>

It is a pathetic process to wade through the vague descriptions of those days; the glimmerings of form relations here and there met with seem to us but very illiberal reward for the patience, industry and conscientiousness which characterized the work of those earlier investigators. Research centered about the "nerve-cells," the "nerve-fibers," and the relations of the latter to the former. The more notable contributions came from Ehrenberg, Purkinje, Valentin, Remak, Schwann; Hannover, Stilling, Helmholtz, von Kölliker, Wagner, Bidder, Gerlach, Schroder van der Kolk and Lockhart Clarke. With Deiters came light. The advent of his "Theorie der centralen Ganglienzelle" marks an epoch in neurology. He distinguished sharply between the protoplasmic processes and the axis-cylinder process of the central ganglion cells. Though foreshadowed by the views of Remak, Wagner and Helmholtz,

<sup>1</sup>Stieda, L.: "Geschichte der Entwicklung der Lehre von den Nervenzellen und Nervenfasern während des XIX Jahrhunderts. I Teil von Soemmering bis Deiters." Festschrift zum siebenzigsten Geburtstag von Carl von Kupffer. Jena, 1899.

the precise, objective descriptions of Deiters may fairly be regarded as by far the most influential in leading up to the modern conception of the nature of the biological unit in the nervous system.

The cell theory was founded in 1838 by Schleiden, and supported and extended in 1839 by Schwann. It underwent later important corrections in the hands of Max Schultze, who developed the protoplasm theory. Virchow's twenty lectures on cellular pathology perhaps did most to inculcate the doctrine of "vital units" in the minds of medical men, making, as he did, the cell the starting point for the investigation of physiological and pathological problems. The world avowedly accepted the cell doctrine for the whole human and animal body; tacitly it did not accept it for the nervous system, for there were always the troublesome fibers. "Nerve-cells" were talked about; they were unipolar, bipolar and multipolar; "nerve fibers" were talked about, and some of them were known to be processes of nerve cells. No precise conception of the extent of a "nerve cell" or of the relation in general of "nerve fibers" to "nerve cells" had yet been arrived at. Ideas were vague; anatomical conceptions were confused.

Neuro-pathology came to the rescue. Out of the observations of Waller and Türck on secondary degenerations came the idea of trophic domains with the nucleated "nerve-cell" as a center. Disease processes affecting the nervous system electively confine themselves to certain definite territories. Von Gudden's experiments on young animals supplemented the studies of secondary degeneration by those of secondary atrophy. The doctrine of system diseases developed. Gowers introduced his conception of the "upper motor segment" and the "lower motor segment." Certain "cells" in the cerebral cortex and the "fibers" of the pyramidal tract correspond to the "upper segment"; the "multipolar ganglion cells" of the motor nuclei of the brain and cord, together with the motor "nerve fibers" of the peripheral cerebrospinal nerves constitute the "lower segment."

Anatomical and embryological studies were progressing. Flechsig was finding that his study of the serial sequence of the medullation of bundles of nerve-fibers in the central nervous

system was isolating tracts of nerve fibers, which corresponded in a striking manner with the degenerative processes met with in elective nervous diseases. His made his epoch-making discovery that the nerve-cells in development are at first devoid of processes; that they wander as independent cell-units from their birthplaces; that after a certain time cell-processes are developed—an axis cylinder process first and protoplasmic processes or dendrites later; that the axis-cylinder processes of all nerve fibers are to be regarded as outgrowths from, and, therefore, as constituent parts of, nerve cells; and, finally, that all connections of a nerve fiber, other than that with the cell-body, which gives it origin, are either indirect or have arisen secondarily. The first fruits of the wonderful Golgi method were being gathered. The sharp black silhouettes of the nerve-cell body and of the complex processes connected with it were beginning to excite attention outside of Italy.

Just at this juncture appeared the brilliant and far-seeing article of Forel.<sup>2</sup>

In "Einige hirnanatomische Betrachtungen und Ergebnisse" he developed in a most forcible manner the doctrine of *the individuality of the nerve elements*; the unit in the nervous system includes not only the "nerve-cell" of the books, but also the processes which depend for their nutrition upon it, including the axis-cylinder process of a nerve fiber, as far as the degeneration can be traced. The limits of a given degeneration in a pathological process correspond in extent to those of the nerve-unit or units involved in the lesion. The papers of Forel and His mark the beginning of the downfall of the old views which separated so sharply the "nerve cells" from the "nerve fibers." The "nerve cell" and the axis cylinder of a "nerve fiber" were seen to represent parts of a single anatomical and physiological unit.

In the years which immediately followed Santiago Ramón y Cajal began to pour forth that steady stream of contributions to the anatomy of the nervous system which has excited the admiration and compelled the gratitude of every one interested in the solving of neurological problems. Golgi had devised

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<sup>2</sup>Forel, A., "Einige hirnanatomische Betrachtungen und Ergebnisse." Arch. f. Psychiat., Berl., Bd. XVIII (1887), S. 162-198.

the method and had led the way to great results. He had described the two main categories of nerve cells (Type I and Type II), discovered the side-fibrils, and made exhaustive studies of the neuroglia. Ramón y Cajal used chiefly the rapid Golgi method. The great value of his work lies in the systematic objective study of a great many different parts of the nervous system by the chrome silver method. Paper followed paper in which, one after another, the spinal cord, the retina, the cerebral cortex, the cerebellum, the corpora quadrigemina, the olfactory lobes, the medulla oblongata and pons, and the sympathetic system were examined and the results yielded by the especial method faithfully recorded. In after years, when the neurological history of these times shall be written, it would seem likely that Ramón y Cajal's name will be most remembered on account of his notable contributions to the special microscopic anatomy of various parts of the nervous system rather than to its general histology. At the time of their appearance, however, these articles attracted more attention from the side of general histology than from that of microscopic anatomy. Ramón y Cajal denied the existence of a diffuse nerve-network in the sense of Golgi, as Golgi before him had denied the existence of a diffuse nerve-network in the sense of Gerlach. He challenged Golgi's ideas of the morphological distinctions between sensory and motor cells. He asserted that in Golgi preparations, except perhaps in the sympathetic system, and in some insects, neither dendrites nor axones could be seen to anastomose with one another—all ended by free terminations in, among, or upon the cell-bodies or branches of other nerve-units. He believed, therefore, from the chrome silver pictures that the nerve-units, aside from the exceptions mentioned, are completely independent of one another. His description of the forms and mode of branching of the units in the most diverse parts are by far the most complete which have been contributed by a single investigator. A large part of his success depended upon his application of the silver method to embryonic tissues and to the tissues of a series of animals. His views were in large part accepted by von Kölliker, von Lenhossék, van Gehuchten, Retzius, Schäfer and

Berkley, all of whom made important contributions along similar lines.

The studies in the comparative anatomy of the nervous system undertaken by Bela Haller, Nansen, Fusari and Retzius lent support to the views above mentioned. The methylene blue method of vital staining introduced by Ehrlich was also confirmatory, at least in many respects.

In 1891 Waldeyer<sup>3</sup> published a collective review of the observations made up to that date and stated as the main results (1) that the axis cylinders of all nerve fibers (motor, secretory and sensory), conducting centrifugally or centripetally, have been proven to proceed *directly* from cells; a connection with a fiber network, or an origin from such a network, does not exist; and (2) that all these nerve fibers end free, with "end-arborization" (Kölliker), without the formation of networks or anastomoses. Combining the two, he suggests that "the nervous system consists of numerous nerve-units (neurons) not connected with one another anatomically or genetically. Each nerve-unit consists of three parts—the nerve cell, the nerve fiber and the end-arborization. The physiological conduction-process can run either in a direction from the cell to the end-arborization or in a reverse direction. The motor conductions run only in the direction from the cell to the end-arborization, the sensory sometimes in the one, sometimes in the other direction." He acknowledges that his ideas of the mode of conduction are based on the view that no anastomosing nerve networks occur, but only a nerve feltwork (neuropilema, His). He states, however, that "if we assume with Golgi and B. Haller the existence of nerve networks, the view is modified somewhat, but we can still retain the nerve-units. The limits between the two nerve-units would then always lie in a nerve net-work and, anatomically at least, be not exactly definable by our present methods."

It is obvious that the essential feature of the whole work up to this time, and this is the point that Waldeyer wishes to emphasize, is the demonstration of the existence in the nervous

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<sup>3</sup>Waldeyer, W., "Ueber einige neuere Forschungen im Gebiete der Anatomie des Centralnervensystems." Deutsche med. Wchnschr., Leipzig, Bd. XVII (1891), S. 1244, 1267, 1287, 1331, 1352.



system of definite *nerve-units*. The view arrived at by the pathologists and embryologists here found its histological support. The nerve-unit (Waldeyer's neurone) consists of three parts, the "nerve-cell," the "nerve fiber" and the "end-arborization." In my opinion,<sup>4</sup> already expressed in several places, this doctrine of the nerve-units (Waldeyer's neurones) is *nothing more or less than the application in full of the cell-doctrine to the nervous system*. The "nerve-cell" of the books is only a part of the *real* nerve-cell. The "nerve-units" of Forel and of Waldeyer are the *complete* nerve-cells. The introduction of the term nerve-unit or neurone was a happy hit; the name spread like wild-fire and with it the important conception of the nature and extent of the unit. Scarcely in any other way could the old confusion as to the cell-unit arising from the use made of the terms "nerve-cell" and "nerve-fiber" have been so quickly done away with. The failure to recognize the cell-body and the axis cylinder process of a nerve fiber as integral parts of a single cell would have been but slowly corrected had we not had a new name. Even now if we teach beginners that these complete units are nerve-cells they are too often puzzled by the text books, nearly all of which write of nerve-cells and nerve-fibers according to the old usage. The suggestion of Schäfer and Sherrington to use the word "perikaryon" to replace the word "nerve-cell," as formerly used, is a good one. Either this word or the term "cell-body" should certainly be employed and the term "nerve-cell" reserved to indicate the whole nerve-unit or neurone. Should this be happily accomplished the terms "nerve-unit" and "neurone" could, if desired, be dropped, for they would then have served their purpose.

Following upon the establishment of the doctrine of nerve-units or neurones came several accessory theories which have been widely diffused and which by a number of workers have come to be regarded as actually a part of the nerve-unit or

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<sup>4</sup>Barker "On the Validity of the Neurone Doctrine." Amer. J. Insan., Balt., 1898-9, vol. LV, pp. 31-49. "The Nervous System and its Constituent Neurones." N. Y., D. Appleton & Co., 1899—"Progress of Neurology," Phil., M. J., Jan. 28, 1899.

neurone doctrine. Among these may be mentioned (1) the theory of the complete independence of the nerve-units from one another except by contact or contiguity; (2) the theory of the ameboid movement of the processes of the nerve-unit or neurone—often known as the “retraction theory,” and (3) various theories concerning the structures in the nerve-unit or neurone which take part in the conduction processes in the nervous system.

As a matter of fact, most of the objections which have been advanced against the neurone doctrine have in reality been urged against one of these accessory theories rather than against the conception of the nerve-unit or neurone *per se*.

*ad. 1.*—The theory of relation by contact only among morphologically completely independent units was built up almost entirely upon microscopic pictures obtained by Golgi's method. As we know now, this procedure, while yielding magnificent results with regard to the principal points concerning the external morphology of the neurone, is insufficient to give information concerning the more minute relations which exist at the points where the terminals of the processes of one nerve-unit come into relation with other nerve-units. It has been established by other methods:—

(a) That coarser anastomoses occasionally occur between the dendrites of different neurones;

(b) That the terminals of the axone and collaterals of one neurone enter into very intimate protoplasmic relations with the perikaryons and dendrites of other neurones (Held's concrescence);

(c) That fibrillary structures can be seen, when the tissue is stained by certain methods, passing from one nerve-unit or neurone into another.

The “contact theory” is still vigorously supported by a number of neurologists, notably by Semi Meyer.<sup>5</sup> The evidence brought forward by Dogiel, Held, Apáthy and Bethe is, however, so strong that the contact-theory or theory of complete morphological independence of the nerve-units or neurones must be considered as having a very doubtful basis, if

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<sup>5</sup>Meyer, S., “Ueber centrale Neuritenendigungen.” Arch. f. mikr. Anat., Bonn, Bd. LIV (1899), S. 296.

not as actually refuted. Considering what we know of the intercellular bridges and protoplasmic fibrils which connect other cells with one another in other parts of the body, it would be surprising if the nerve-cells or neurones were not similarly connected. As will be recalled from the quotation from Waldeyer cited above, that anatomist did not make the doctrine of the nerve-units or neurones contingent upon absolute histological independence, but stated that "even if networks or anastomoses exist we can still retain the nerve-units." In this connection the lucid statement of Carl Weigert,<sup>6</sup> whose sound judgment many will be willing to trust, may be recalled. He says: "The interesting doctrine of the independence of the 'neurone' has nothing to do with the doctrine of free terminations. This is simple enough, since the doctrine of the neurones, although not under this excellent name, long before Golgi's method was thought of, was well known in pathological anatomy. The whole doctrine of the typical secondary degenerations rested entirely upon what we now call the neurone doctrine. We knew long ago that on destruction of the central gyri or any other part of the pyramidal tract, only those parts of the pyramidal tract degenerated which were cut off from their cell-bodies of origin. We knew long ago that the motor nerves stand under the trophic, or, as it is now called, idioplastic, influence of the cells of the ventral horns only, the sensory under that of the spinal ganglion cells, and that all these nerve-tracts, and no others, degenerate after separation from their idioplastic center.

"The system of any idioplastic (trophic) center, with its territory, is, however, exactly identical with one neurone. Whether the processes of two neurones adjoin one another directly, or are separated from one another by a small interspace, may have a slight anatomical interest; biologically it has none."

*ad 2.*—The so-called "retraction-theory" may be dismissed with a word. It never had a basis in observation or experi-

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<sup>6</sup>Weigert, C.: "Die histologische Technik des Centralnervensystems." Merkel-Bonnet's "Ergebn. d. Anat. u. Entwicklungsgesch.," Wiesbaden, Bd. V (1895), p. 28.

ment, for the note of Wiedersheim can scarcely be regarded as such. With the demonstrations of the intimate relations between neurones brought by the preparations of Held, Apáthy and Bethe all plausibility to the theory disappears. The only wonder is that a theory with so little objective support should have been so widely diffused and so extensively employed for the explanation of physiological and pathological conditions.

*ad 3.*—Coincident with the variation of views concerning the minute internal structure of the nerve-cell protoplasm, a number of theories as to the physical basis for conduction have originated. Max Schultze<sup>7</sup> had asserted that the axis cylinder and the ganglioncell possessed a fibrillary structure, and there early developed a view that these fibrils represent the conducting substance. Nissl believed that the "unstainable substance" in his methylene blue preparations must be regarded as the functionally essential part of the cell. Held thought that the whole protoplasm might conduct, but did not deny that the longitudinal beams of the protoplasmic meshwork might offer channels of lower resistance to conduction than others.

An enormous stride forward has, however, been made by Stefan Apáthy<sup>8</sup> of Kolozsvár through the introduction of new technical methods and thorough application of them to the nervous tissues of certain animal types. The distinguished Hungarian investigator began these studies as early as 1884, and since then has made a number of publications, the most complete article being that published from Naples in 1897.

A succinct epitome of his researches and their general bearings was given by Apáthy<sup>9</sup> in his address before the International Congress of Zoology in Cambridge in 1898. His demonstration of the neurofibrils and their relations to the nerve-units represents the most important discovery in neuro-

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<sup>7</sup>Schultze, M.: Article in S. Stricker's "Manual of Histology," American transl., 8vo, New York, 1872.

<sup>8</sup>Apáthy, S.: "Studien ueber die Histologie der Najaden." Math.-naturw. Abth. d. ung. Acad. d. Wissensch., 1884, "Das leitende Element des Nervensystems und seine topographischen Beziehungen zu den Zellen. Mittheil. aus der Zool. Station zu Neapel." Bd. XII (1897), H. 4, S. 495-748.

<sup>9</sup>Apáthy, S.: "Ueber Neurofibrillen." "Proc. Internat. Congr. Zool.," Cambridge, 1898, pp. 125-141.

logical anatomy in recent years. I<sup>10</sup> have so recently reviewed the principal features of Apáthy's work that it will be unnecessary to go at this time into its details. Apáthy's methods demonstrate histologically the "neurofibrils" and their constituent "elementary fibrils" most exquisitely; to see his specimens is to be convinced of the existence of these structures as most definite morphological entities. The elementary fibrils form networks in the perikaryons of nerve-cells; there is no beginning or end to the fibril system, it is a continuum. Fibrils may even exist outside the nerve-cells forming a mesh-work—his so-called *Elementargitter*—in invertebrates.

In addition to his demonstration for the first time of the neurofibrils in invertebrates, Apáthy has brought all the proof that can be adduced from the morphological side alone that the neurofibrils represent the conducting element in the nervous system. In his Cambridge address he summarizes the evidence in favor of the conducting nature of the neurofibrils as follows:

- (1) The specificity of the neurofibrils.
- (2) The continuity of the neurofibrils.
- (3) The behavior of the neurofibrils in the ganglion cell.
- (4) The entrance, and, as a matter of fact, the exclusive entrance, of the neurofibrils into the sense cells and their specific topographical relations in the body of the sense cell.
- (5) A penetration of neurofibrils into the depth of the muscle fibers and their specific topographical relations to the sarcoplasm.
- (6) The behavior of the neurofibrils in the gland cells.
- (7) In the cells which form the wall of vessels devoid of muscle.
- (8) The relations of the neurofibrils to various other epithelial cells.
- (9) The extra-cellular continuation of the neurofibrils, the branchings and reciprocal relations of the same to the center.
- (10) The same to the periphery.
- (11) The nerve paths consist in places exclusively of neurofibrils.

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<sup>10</sup>In "The Nervous System and Its Constituent Neurones." N. Y., 1899.

(12) The intimate relation between the length of the distance traversed by a neurofibril in a certain specific cell variety and the developmental stage of the organ, the chief constituents of which are those specific cells.

Apáthy's work abounds with objective descriptions of the topographical relations of the neurofibrils to the cells and outside the cells in the organism. The above researches alone would suffice to indicate the great advance made by this investigator, but in addition his various articles contain a mass of details concerning the finer anatomy and histology of the nervous system and of the sense organs of several types of animals, especially of the leeches. His studies of the neurofibrils in *Lumbricus* and in the *Hirudineæ* are among the most important parts of his work.

It is pleasing to be able to report that Apáthy's views do not in any way conflict with the cell doctrine. His objection to the neurone doctrine is evidently based upon the opinion that it includes also the theory of contact relation or the doctrine of completely isolated units. In a personal communication recently received he states his position as follows: "The neurofibrils are a specific cell product and are distributed originally everywhere only with the processes of these cells; besides, these processes later consist for long distances and at their extremities only of neurofibrils and interfibrillar (or perifibrillar) substance. My opposition to the neurone theory consists solely in this, that I can demonstrate that these processes with their neurofibrils also pass through other cells or that the neurofibril reticula (*Neurofibrillengeritter*) of different cells are connected with one another by means of neurofibrils; that therefore the neurones are not isolated units either in an anatomical or in a functional respect. What there is left over of the neurone doctrine is common to it and the cell doctrine in general; and that is the trophic and other regulatory influence of each definite domain, the center of which is the body of a ganglion cell and within which occur the constituents of the nervous system belonging genetically to the ganglion cell. This I have never denied, but it is simply the cell doctrine; there is nothing specific for the neurones."

Other investigations on neurofibrils have been made by

Becker, Dogiel, Mann, Kronthal and Bethe. The results of the last-named histologist<sup>11</sup> are among the most interesting. Acquainted with the researches and methods of Apáthy, Bethe has undertaken the study of the vertebrate and human nervous system with highly satisfactory consequences. By a specially modified method he is able to differentiate the neurofibrils in human tissues in a manner surpassing by far anything hitherto attained. In the main his findings agree with those of Apáthy, the chief differences being that Bethe does not find the inner network of fibrils in the cell-body, and that he thinks the *Elementargitter* is not "diffuse." If the neurofibrils are the true conducting element in the nervous system, the findings of Bethe with regard to their distribution in the dendrites is interesting as refuting the theory of exclusively cellulipetal conduction in these processes. In Bethe's specimens not all the fibrils entering the cell body by way of the dendrites pass out by way of the axone; many pass out by way of another dendrite. Fibrils can even be followed along one branch of a dendrite into another branch of the same process without entering the perikaryon at all.

Strongly in favor of the view that the neurofibrils are the conducting elements is the observation of Bethe and Mönckeberg<sup>12</sup> in which they make out that at the nodes of Ranvier all constituents of the nerve-fiber are interrupted except the neurofibrils. The perifibrillar substance is absent here, they assert, just as is the myelin sheath and the neurilemma.

Bethe's standpoint with regard to the neurone doctrine is easily understood. He denies the existence of histologically completely independent units, but retains the name for all the parts easily demonstrable as standing in connection with one ganglion-cell body.

Of the more important of the recent reviews of the neurone

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<sup>11</sup>Bethe, A.: "Die anatomischen Elemente des Nervensystems und ihre physiologische Bedeutung." Biol. Centralb., 1898, S. 843.

<sup>12</sup>Bethe, A.; u. Mönckeberg. "Die Degeneration der markhaltigen Nervenfasern der Wirbelthiere unter hauptsächlichlicher Berücksichtigung des Verhaltens der Primitivfibrillen (Zugleich ein Beitrag zur Kenntniss der normalen Nervenfasern)." Arch. f. mikr. Anat., Bonn, Bd. LIV, S. 135.

doctrine may be mentioned those of Adolf Meyer;<sup>13</sup> von Lenhossék;<sup>14</sup> Hoche;<sup>15</sup> Nissl,<sup>16</sup> and Edinger and Wallenberg.<sup>17</sup> The conceptions of the neurone doctrine entertained in these different articles vary somewhat, but all but Nissl retain the conception of the nerve-units. Nissl, in the article in which he develops his theories concerning the gray matter, violently throws the neurone doctrine overboard, maintaining that it is utterly refuted. He appears to see in it only the contact-theory and to have misconceived or lost sight entirely of the real significance of the doctrine of nerve-units. Edinger, on the other hand, as might have been expected, grasps the situation very clearly. To quote from him:

"The neurone theory was deducible in the first place from the results of degeneration pictures (Forel). Indeed, it was long silently accepted by the pathologists before we had the word neurone, the pathologists having recognized that injury of any given nerve or of any cell is never followed by more severe and more direct consequences, beyond a certain domain. It has been customary to look upon this domain as dependent upon the cell nucleus of a definite ganglion cell and then to view the whole unit as trophic. When the proof was brought, chiefly by S. Ramón y Cajal's investigations, that such units could also be distinguished anatomically, the anatomists were justified also in becoming very much interested in the conception that the whole nervous system might be built up of a series of superimposed cell units. From many sides findings which spoke in favor of this were reported, justifying the conception to which Waldeyer, after reviewing all the facts, gave the name of the neurone theory. Whether the ends of

<sup>13</sup>Meyer, A.: "Critical Review of the Data and General Methods of Modern Neurology." *J. Comp. Neurol.*, Granville, Vol. VIII (1899), Nos. III and IV.

<sup>14</sup>v. Lenhossék, M.: Review of Bethe's work in *Neurol. Centralbl.* Leipzig, Bd. (1899), Nos. 6 & 7.

<sup>15</sup>Hoche, A.: "Die Neuronlehre und ihre Gegner." Berlin, 1899, S. 1-52.

<sup>16</sup>Nissl, F.: "Nervenzellen und graue Substanz." *Münch. med. Wchnschr.*, Bd. XLV, 1898, S. 988; 1023; 1060.

<sup>17</sup>Edinger, L., and A. Wallenberg: "Bericht ueber die Leistungen auf dem Gebiet der Anatomie des Centralnervensystems während der Jahre, 1897 und 1898." *Schmidt's Jahrbücher*, Leipzig, Jahrg. 1899. Bd. CCLXII, S. 65.



the neurone are free, as the majority supposed, or whether here and there or even always genuine fusions with neighboring nerve units occur, have been much discussed questions. The majority have, as is well known, declared in favor of a free splitting up of the cell processes around the body or the processes of other cells. But at the time of this report we have learned through Held, Golgi and others that at the periphery of the cell much more complicated relations can be found than correspond to so simple a view. It appears to us as of secondary importance whether one can or not to-day demonstrate everywhere the anatomical limits of the nerve domain dependent on the single ganglion cell, as long as no fact is brought forward which interferes with the conception of the neurone as a biological unit. Such a fact does not exist, however. Indeed, all the pathological relations known at present when the ganglion cell and its processes degenerate can only be explained when one stands on the platform of the neurone theory, and this conception is not at all interfered with by the newly-won pictures of fibrils. We have, however, made a great step in advance in the direction that we now know how inside a single cell fibers out of other cell domains unite with others which form networks in the cell itself, etc.; how the cell often is only a transfer station for fibers of different origin. A separation in the old sense that each cell exists anatomically for itself alone, entering only externally in relation to neighboring cells, can probably be no longer held without modification. But we must all the more hold fast to the fact that the ganglion cell (including all fibers going into it) forms a biological unit. One can easily conceive that in such units the collection and new distribution of paths of the most different origin may take place without on that account giving up the idea of the unit itself. The establishment of the neurone concept was an important fact. It has made many pictures hitherto confusing easily explicable. It has been unusually heuristic in its effects and has permitted us to understand for the first time certain parts of the nervous system, such as the retina, the bulbus olfactorius, the spinal ganglia and the peripheral sense cells. If it is to be given up to-day it must above all be shown that what is known is irreconcilable with it. This has not been shown."

In conclusion I may be permitted again to emphasize my own view. The formulation of the neurone doctrine was the equivalent of applying the doctrine of cell-units to the nervous system. The actual application to the nervous system at so late a date of a doctrine long known and generally appreciated for the other tissues of the body is explicable by the fact that it has been only recently that it has been possible to replace the mistaken conception of the extent of a nerve-cell formerly held by the true conception. If the term "nerve-cell" called up in mind the idea which we now hold of the biological unit in the nervous system there would be no necessity for the use of the word "neurone." But until the textbooks have been expurgated and the nomenclature employed by teachers and writers becomes more precise, some word other than "nerve-cell" would seem necessary. Any one who has the idea of "contact-relation" inseparably connected in mind with the term "neurone" would do well to give up the use of the word and to use "nerve-unit" or "complete nerve-cell" in its place. But for those who have never considered the theory of contact relation as a necessary part of the neurone doctrine, but have regarded the neurone as the unit in the nervous system corresponding to the cells in other parts of the body, the term can be indefinitely retained and even employed with reverence on account of its historical significance.

- 99 "NEUE BEOBSACHTUNGEN ÜBER DIE VERÄNDERUNGEN DER PYRAMIDENRIESENZELLEN IM VERLAUF DER PARAPLEGIEEN" (New Observations on the Changes of the Pyramidal Giant Cells Occurring in Paraplegia). G. Marinescu (Deutsche med. Wochenschrift, May 31, 1900, p. 351).

Marinescu has examined the Rolandic area in six cases of compression of the spinal cord with descending degeneration of the pyramidal tract. The duration of the lesions varied from five months to two years. He found alteration of the cells of Betz in all six cases. Where the spinal lesion had been of short duration the cell-body was swollen, rounded, and presented chromatolysis, chiefly central, and some displacement of the nucleus. In cases of longer duration the lesions were more pronounced, and cellular atrophy was observed. The alteration of Betz's cells occurred from a lesion of the pyramidal tract at any level, but it was greater the nearer the lesion was to the cell-body.

In the study of the motor cortex in a case of amyotrophic lateral sclerosis, Marinescu found great alteration of the giant cells. Most of these cells had disappeared, and the few remaining ones were very atrophic and showed chromatolysis and displacement of the nucleus. Marinescu leaves it undecided whether these cellular changes were primary or secondary.

SPILLER.

(b) THE PATHOLOGICAL CHANGES IN THE NEURONE  
IN NERVOUS DISEASE.<sup>1</sup>

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In discussing the pathological changes in the neurone in nervous disease the most important question we have to answer is: Does pathology afford any support for the neurone doctrine—*i. e.*, are the changes that occur in disease of the nervous system of such a character that we may obtain from them evidence of the existence of neurones?

To answer this question it must be shown whether degenerative processes have a tendency to affect distinct groups of nerve cells, including their dendritic processes, axones, collaterals and terminal ramifications, to the partial or complete exclusion of other groups.

It must be shown whether the neurone in its entirety, either motor or sensory, is affected by disease—*i. e.*, whether the cell-body may become diseased and die without disease and death of the axone, or whether the axone may suffer or perish without destruction or involvement of the cell-body.

It must be shown whether a morbid process is always limited to a central or peripheral neurone, or whether degeneration passes from one to the other, from a motor to a motor neurone, or from a sensory to a sensory neurone, without any interference at the parts that have been regarded as points of contact of the neurones. It must be shown whether disease of a sensory neurone affects a motor neurone, or *vice versa*. To put the whole matter in a few words, we must obtain from pathology the evidence that what we recognize as a neurone suffers when diseased in so peculiar a manner that we may recognize in this involvement the individuality of the structure.

I shall not give a résumé of the pathology of the cell-body; that has been well done by capable investigators—van Gehuch-

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<sup>1</sup>From the William Pepper Clinical Laboratory (Phœbe A. Hearst Foundation). Read at the twenty-sixth annual meeting of the American Neurological Association, May 1, 2, and 3, 1900.

ten, Marinesco, Ewing, Barbacci, Robertson and others; but I shall endeavor to apply some of the literature on the subject and the results of my own investigations to the solution of the problems I have mentioned.

We cannot deny that degeneration has a very evident tendency to involve distinct groups of nerve-cells, including their various processes. The existence of systemic diseases and of combined systemic diseases admits of little dispute, and we have in this limitation of morbid processes one argument in favor of neurones. Tabes, amyotrophic lateral sclerosis, Friedreich's ataxia and other diseases might be mentioned in illustration. It is not because all the axones affected in these diseases conduct impulses in one direction that they degenerate. We have in tabes, for example, a degeneration of posterior root fibers, including in some cases the descending roots of certain cranial nerves that are similar to the posterior roots of the spinal cord—the sensory fifth and the combined root of the ninth and tenth nerves—as I have seen in four or five cases. We do not have as a common finding in tabes degeneration of other ascending tracts; of the direct cerebellar or of the fillet. We have a selective degeneration of the primary sensory neurones, and a degeneration that does not as a rule pass to sensory neurones of another system. We have manifested in the pathological changes of certain systems an evidence of an individuality of these systems. I shall not dwell on this aspect of the subject lest I infringe on Dr. Sachs' rights.

We may inquire to what degree a neurone in its entirety becomes diseased. If we cut an axone, either central or peripheral, we know from the investigations of Nissl, Marinesco, van Gehuchten and many others that the cell-body in which this axone arises undergoes rapid alteration and possibly death, and that this alteration is probably more intense the nearer to the cell-body the division of the axone is made. Not only is the cell-body affected, but the central portion of the axone may also suffer more or less alteration. I need not refer to the degeneration of the peripheral end; that is well recognized as the degeneration of Waller. The earlier in the life of the animal the injury to the axone occurs the greater is the atrophy of the cell-body in which the axone arises. In a case of Pott's

disease that I<sup>2</sup> studied the compression in the cervico-thoracic region probably began at the third year of the child's life, and by the eighth year the direct cerebellar tract *below* the compression and the cell-bodies in the column of Clarke had disappeared. I attach much importance to these findings, as I believe that mine was the first case recorded of complete atrophy of the cells of Clarke's column from a cervico-thoracic lesion.

Barker<sup>3</sup> in 1897 found *chromatolysis* in the cell-bodies of Clarke's columns in epidemic cerebrospinal meningitis, and explained it as a "reaction at distance."

Sano<sup>4</sup> in July, 1897, described *chromatolysis* of the cell-bodies of the column of Clarke, resulting from the pressure of a tumor on the direct cerebellar tract, the compression being chiefly at the fourth cervical segment. The alteration in Clarke's column consisted of central chromatolysis and some deformity of the nucleus. From the study of this case Sano concluded that the law of reaction at distance in a cell-body as a result of injury to its axone exists as truly for the central neurones as for the peripheral.

In my paper read in January, 1898, at the Johns Hopkins Hospital and published in June, 1898, I described complete destruction of the cell-bodies in Clarke's columns, and not merely chromatolysis, from a cervico-thoracic lesion.

Van Gehuchten<sup>5</sup> also has observed chromatolysis of these cell-bodies after the division of the spinal cord in the dog.

Sano,<sup>6</sup> in a later paper, reported the finding of chromatolysis in Clarke's column in six cases, resulting from lesions of the direct cerebellar tract.

I have become somewhat skeptical as to the value of chromatolysis in Clarke's column, and am inclined to think that these cells are especially liable to react in this way from many causes.

The change in the central end of the motor axone, apart from chromatolysis, after division of the axone has occurred, is

<sup>2</sup>Spiller, The Johns Hopkins Hospital Bulletin, No. 87, June, 1898.

<sup>3</sup>Barker, Brit. Med. Journal, 1897, Vol. II, p. 1839.

<sup>4</sup>Sano, Journal de Neurologie, July 20, 1897, p. 276.

<sup>5</sup>Van Gehuchten, Journal de Neurologie, June 5, 1898, p. 238.

<sup>6</sup>Sano, Journal de Neurologie, Aug. 5, 1898, p. 313.

not rapid, especially when the injury is at some distance from the cell-body. I have observed retrograde atrophy of the pyramidal fibers in several instances, but only when the transverse lesions were of long duration.

I have studied a case of peripheral nerve degeneration, the symptoms of which had existed for at least five or six years.

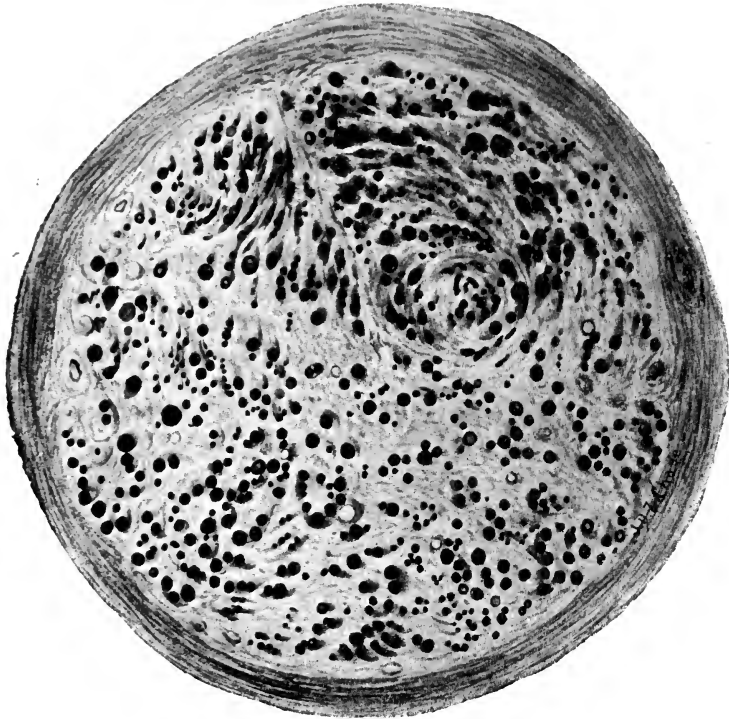


Fig I. Case of chronic neuritis of five or six years' duration. Nerve fibers of the left internal plantar nerve showing much degeneration. Some of the cell-bodies, from which the axones of this nerve arise, show the "reaction at distance" in the form of central chromolysis and displacement of the nucleus.

The terminal portions of some of the branches from the sciatic show very distinct degeneration of the nerve fibers and overgrowth of the connective tissue in and about the nerve bundles. (Fig. I.). Many of the cell-bodies of the anterior horns of

the lumbar region present central chromatolysis and displacement of the nuclei (Fig. II.), and this is in a case of chronic neuritis.

Nissl's stain is, of course, the most valuable for the study of nerve cells, but pronounced degeneration may often be told by the ammonium carmine stain. In my case of Pott's disease, to which I have referred, and in a case of acute poliomyelitis in an adult,<sup>7</sup> the destruction of cell-bodies was most evident by the carmine stain, and I am somewhat skeptical as to the importance of cellular changes that have existed many years, and cannot at all be detected by the carmine stain.

It seems that in acute diseases involving the axone, the cell-body in which the axone arises suffers, but if the disease

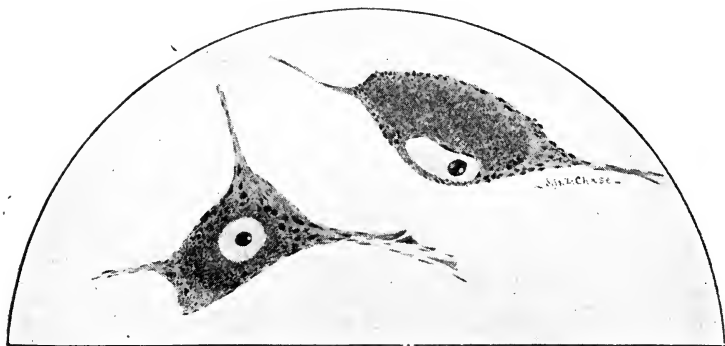


Fig. II. Chronic neuritis of five or six years' duration. The cell-bodies in the anterior horns in the lumbar region show the "reaction at distance."

becomes chronic, and is in the distal part of the axone, the cell-body may not show any serious alteration, even by Nissl's stain. The neurone as a whole suffers, but the chromatolysis may disappear, unless the degeneration involves a portion of the axone too near the cell-body. Dejerine and Thomas,<sup>8</sup> in a case of neuritis, found the nerves greatly altered without any appreciable change by the Nissl method in the cell-bodies in

<sup>7</sup>Sherman and Spiller, *The Philadelphia Medical Journal*, March 31, 1900.

<sup>8</sup>Dejerine and Thomas, *Comptes rendus de la Soc. de Biologie*, 1897.

which these fibers arose. Cellular alteration, possibly, had occurred in this case, and the cell-bodies had recovered. The importance of chromatolysis is disputed. To Dejerine and Thomas it has little value, but to Schaffer it is an index of degeneration of a more essential portion of the cytoplasm. Dejerine<sup>9</sup> showed in 1889, by the examination of nine cases of tabetic muscular atrophy, that the peripheral nerves in their terminal portions were much diseased, and yet no cellular changes were found in the spinal cord. The Nissl method, of course, was not employed. If chromatolysis occurred in the cell-bodies in these cases—as it did occur in my case of chronic neuritis, the material from which I received from Dr. Mills;

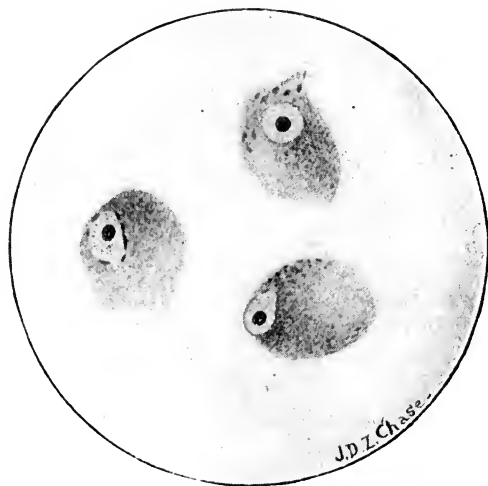


Fig. III. Cell-bodies of the facial nucleus in a case of peripheral facial palsy of six months' duration. A few of the cell-bodies within the nucleus are in a condition of almost complete chromatolysis, with displacement of the nucleus.

and in a case of facial paralysis of six months duration, now reported by Dr. Mills and myself (Fig. III.)—it had at least not led to atrophy of the cell-bodies.

Van Gehuchten<sup>10</sup> holds that the restoration of a cell-body

<sup>9</sup>Dejerine, "Sur l'atrophie musculaire des ataxiques," 1889.

<sup>10</sup>Van Gehuchten, "Anatomie du système nerveux de l'homme," third edition, Vol. I, 1900, and Presse méd., 1899, p. 3.



from chromatolysis occurring after division of the axone, is not dependent upon the reunion of the axone, and he refers to a similar statement made by Nissl. Van Gehuchten believes that reunion of the axone is without effect upon the restoration of the chromophilic bodies, but if restoration of the axone does not occur, the cell-body finally atrophies and disappears. Marinesco<sup>11</sup> advances somewhat different views. According to him the reparative process in a cell-body after its axone is cut is dependent upon the reunion of the axone, although both Marinesco and van Gehuchten seem to be in accord as to the final atrophy of the cell-body when the two ends of the axone remain ununited. Simple chromatolysis seems to me to be of doubtful value as indicative of cellular degeneration if, as van Gehuchten believes, it disappears, although the cell may die later, and is only a temporary reaction of the cell-body after division of its axone. Van Gehuchten has been unable to obtain even temporary chromatolysis in the spinal cell-bodies of rabbits and dogs by cutting the axones of these cell-bodies, but with de Buck,<sup>12</sup> he has found chromatolysis in the spinal cord of man after amputation of a limb. Chromatolysis in man after amputation of a limb has been observed also by Flatau<sup>13</sup> and by Sano.<sup>14</sup> I have had the rare good fortune to obtain the spinal cord from a case in which amputation at the hip joint was followed by death after five and a half days. The cell-bodies in the lumbo-sacral region on the side of the amputation have undergone great chromatolysis, and in some of the cell-bodies displacement of the nucleus has occurred. The cell-bodies in the anterior horn of this side stain very faintly. On the other side of the cord the cell-bodies of the anterior horn are much more deeply stained, although some of them show chromatolysis. The difference in the condition of the cell-bodies of the two horns is striking. The chromatolysis in many cell-bodies is complete; in others, it is merely central.

The neurone in all its parts is not always promptly affected when the cell-body is seriously diseased. The case of acute

<sup>11</sup>Marinesco, *Presse méd.*, 1898, pp. 201-206.

<sup>12</sup>Van Gehuchten and de Buck, *Journal de Neurologie*, 1898, p. 94.

<sup>13</sup>Flatau, *Deutsche med. Wochenschrift*, 1897, p. 278.

<sup>14</sup>Sano, *Journal de Neurologie*, 1897.

poliomyelitis reported by Dr. Sherman and myself (*l. c.*), is an evidence of this. The disease was of short duration, but was sufficiently long to cause destruction of the cell-bodies of the anterior horns, and to leave the motor roots unaffected, not even causing swelling of the axones. I could refer to a number of similar cases in literature. In the case of hemorrhagic pachymeningitis reported to this Association last year by Dr. McCarthy and myself,<sup>15</sup> intense alteration of the cell-bodies was observed, but none of the axones. If the objection should be raised that this cellular change was recent, and possibly from edema, and therefore alteration of the axones had not time to develop, I would reply that precisely the same changes were observed by Hirsch,<sup>16</sup> in a case of amaurotic family idiocy, and that this observation by Hirsch and the improbability of an edema of *recent* origin in our case, would lead me to believe that the cellular changes throughout the central nervous system probably were not recent. The changes in Hirsch's case could not have been of post-mortem origin, as the necropsy was made four hours after death. In his case, however, the change in the cell-bodies was associated with degeneration of the pyramidal tracts, and in ours it was not. I have recently studied another case in which the same cellular changes existed, and degeneration of the pyramidal tracts did not occur. This was a case with the symptoms of meningitis in a feeble-minded child. Cellular changes such as those just described I have been unable to find in cortical edema.

It would seem, therefore, that the cell-body, though greatly altered morphologically, is still capable of exerting an influence on the health of the axone, and that so long as the cell-body persists and is not too seriously altered, the axone may live. An axone is only partially dependent upon the cell-body for its vitality, and if the blood supply to it at a considerable distance from the cell-body is cut off, that portion deprived of the circulation dies, but the cell-body seems to be necessary to the

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<sup>15</sup>Spiller and McCarthy, THE JOURNAL OF NERVOUS AND MENTAL DISEASE, 1899, p. 677.

<sup>16</sup>Hirsch, THE JOURNAL OF NERVOUS AND MENTAL DISEASE, 1898, p. 538.

axone to enable it to appropriate the nourishment brought to it.

Diseases that are due to poisons also have a decided tendency to affect certain neurones in their entirety, as was evident in the case of Landry's paralysis reported by Dr. Mills and myself,<sup>17</sup> in which only the peripheral motor neurones were affected. Similar cases are reported in literature.

Central motor neurones degenerate in a manner very like that of the peripheral motor neurones. Marinesco<sup>18</sup> found very intense alteration of the cells of Betz, and only of these cells, in the paracentral lobule, following a lesion of the internal capsule. These large cell-bodies may entirely disappear, as the result of such a lesion. The cellular changes consist of tumefaction of the cell-body, chromatolysis and alteration and displacement of the nucleus. Marinesco thus confirms the statement of v. Monakow,<sup>19</sup> that the large pyramidal cell-bodies of the paracentral lobule disappear after destruction of the internal capsule. From the integrity of the other cell-bodies of the paracentral lobule, Marinesco seems to conclude that these smaller cell-bodies do not give origin to motor fibers. His words in speaking of the cells of Betz are: "*Ce sont ces cellules qui donnent naissance aux fibres pyramidales.*" As the large cells of Betz are scarce in the lower portions of the motor cortex, we may hesitate to accept the view that Betz's cells are the only ones in the motor cortex that are motor in function. Marinesco refers to the fact that Dotto and Pusateri have seen cellular changes in the motor cortex following destruction of the internal capsule.

Ballet and Faure<sup>20</sup> found that after the fibers of projection from the motor area were cut, the motor cortical cell-bodies atrophied. The cellular changes were similar to those that occur in the motor cell-bodies of the cord when the peripheral motor fibers are divided. They experimented on seven dogs, and presumably made the division of the fibers not far below

<sup>17</sup>Mills and Spiller, *THE JOURNAL OF NERVOUS AND MENTAL DISEASE*, 1899.

<sup>18</sup>Marinesco, *Revue neurologique*, 1899, p. 358.

<sup>19</sup>Von Monakow, "*Gehirnpathologie*," p. 118.

<sup>20</sup>Ballet and Faure, *Semaine méd.*, 1899, p. 109.

the cortex. The point of attack is of importance in the production of cellular lesions, as the nearer the interruption of the axone is to the cell-body, the greater is the alteration of the cell-body.

I have studied the paracentral lobule in five cases of capsular lesion, three of which were due to hemorrhage of various durations, but I have been unable to convince myself that characteristic changes occurred in the cells of Betz. I have been more fortunate in my examination of the cortex in cases of long duration. One of the brains studied was from a half-grown boy who had survived for ten or eleven years the closure of the left Sylvian artery. The motor area was destroyed except in its extreme upper portion. The motor axones from this area were cut near their origin. Very few cell-bodies were found in the paracentral lobule, and the cells of Betz were almost entirely absent. The sections from the paracentral lobule of this brain were exceedingly interesting in comparison with those from the paracentral lobule of a brain in which the lower part of the left motor area was destroyed by porencephaly. The motor axones above this lesion were not injured so close to the cell-bodies. The porencephaly naturally suggests a congenital lesion, and the motor axones from the upper left motor area were probably arrested in their downward growth. The results were similar to those produced by dividing the axones. As the lesion was not so near the cell-bodies of origin in the paracentral lobule as in the former case, fewer cell-bodies had disappeared entirely, and yet those present were much below the normal number, were small and rounded, the pericellular spaces were large, and the cells of Betz seemed to be absent. These two cases have given results very like those obtained experimentally by Ballet and Faure, only the cellular changes in my cases were much more intense. Destruction of the cells of Betz from cerebral lesions have been observed not only by v. Monakow, but also by Moeli, Henschen, and Mahaim.

The peripheral sensory neurone reacts in a peculiar way to traumatism. It is very remarkable that, while division of the peripheral process of a spinal ganglion cell-body causes very distinct degeneration of this cell-body (Lugaro, Mering, Flem-

ing, van Gehuchten, Cassirer), and even complete destruction of the cell-body according to some, division of the central process causes no cellular reaction. This is a very interesting statement and of much value in the explanation of tabes. Marinesco<sup>21</sup> denies that the cell-body of the peripheral sensory neurone dies when its peripheral process is cut, as van Gehuchten believes.<sup>22</sup>

Cassirer<sup>23</sup> has found that after a peripheral nerve is cut a large number of cell-bodies in the spinal ganglion belonging to this nerve degenerate, but only a few are so greatly altered that they probably undergo complete destruction. As a result of this cellular degeneration he finds that a moderate degeneration of posterior roots occurs. The changes which Cassirer found in the posterior roots as a result of the division of the peripheral nerve were slight, as they were also in some similar experiments performed by Redlich, and therefore Cassirer thinks his findings afford no support for the theory that tabes begins in the peripheral nerves. There is no comparison, he thinks, between the slight degeneration of the posterior roots when a whole nerve is cut and the intense degeneration of these roots in tabes.

In a Gasserian ganglion removed recently by Dr. Harvey Cushing for trifacial neuralgia I found alteration by Nissl's stain in quite a number of the cell-bodies of the ganglion—by no means the majority, however—but was not able to detect any change in the sensory root, although I found alteration of the sensory root in another ganglion removed by Dr. Cushing. Krause<sup>24</sup> also reports disease of a sensory root. My examination of the ganglion referred to and of its sensory root has given results similar to those obtained by Cassirer and Redlich in experiments on the spinal ganglia, *i. e.*, alteration of cell-bodies of the ganglion with little alteration of the cellulifugal processes.

The destruction of the spinal ganglion was very much less

<sup>21</sup>Marinesco, *Presse méd.*, 1898, p. 201.

<sup>22</sup>Van Gehuchten, "*Anatomie du système nerveux de l'homme*," third edition, Vol. I, 1900.

<sup>23</sup>Cassirer, *Deutsche Zeitschrift für Nervenheilkunde*, Vol. 14, p. 150.

<sup>24</sup>Krause, "*Die Neuralgie des Trigeminus*," Leipzig, 1896.

in Cassirer's experiments than in those of Lugaro and van Gehuchten, and therefore we might expect slight alteration of the posterior roots in Cassirer's cases. It seems impossible to examine a picture in which the cells are as few in number as in that given by van Gehuchten<sup>25</sup> on p. 335 of the recent edition of his text book, and to believe that the cellulifugal processes of the cell-bodies of the ganglion in such a case were only slightly degenerated.

Schaffer's<sup>26</sup> finding of normal cell-bodies by the Nissl method in the spinal ganglia in tabes is a further proof that the cell-bodies of the spinal ganglia do not react promptly to lesions of their central processes. I also in the examination of a case of tabes by Nissl's stain have found the cell-bodies in the spinal ganglia of the lumbar region normal.

We see, therefore, that on the whole there is a tendency for the entire neurone to undergo alteration when it is attacked by disease or injured experimentally, but that a portion may be more resistant than the rest and may recover even though at first affected, although the end in union with the cell-body may finally atrophy. The earlier in life the lesion occurs the less is the resistance of the neurone. From these facts we have some evidence of the individuality of the neurone.

A very interesting question is in regard to the degree of limitation of degeneration to one set of neurones, either sensory or motor. It is quite certain that a restriction exists, but occasionally this restriction is overcome. Tertiary degeneration—and by that I mean a degeneration in another series of neurones resulting from secondary degeneration of a series of neurones nearer the origin of the impulses—does not seem to be of very frequent occurrence. It is important to make a distinction in regard to this matter. The changes that occur in a second neurone associated with *primary* degeneration in a first neurone—and by that I mean degeneration not due to a focal lesion—cannot be compared with the degeneration of one neurone following degeneration of another from a focal lesion. In the first instance we have reason to believe that the first neu-

<sup>25</sup>Van Gehuchten, "Anatomie du système nerveux," third edition, Vol. I.

<sup>26</sup>Schaffer, Neurologisches Centralblatt, 1898, p. 2.

rone that undergoes primary degeneration does so as a result of imperfect development, or possibly as the result of the action of a poison, and we do not know that the same causes have not produced the degeneration of the second neurone. In amyotrophic lateral sclerosis, for example, of which I have studied the specimens of two cases, we have no proof that the degeneration of the peripheral motor neurones is the result of degeneration of the central motor neurones, or *vice versa*.

I have examined cases of hemiplegia in the adult with sclerosis of the pyramidal tract, but have never seen degeneration of the cell-bodies of the anterior horns by the carmine stain and of the nerve fibers arising from them, *i. e.*, I have not found tertiary degeneration in these cases. In my case of Pott's disease of early development, to which I have referred, in which both pyramidal tracts were destroyed, the cell-bodies in the anterior horns in the lumbar region by the carmine stain were normal in shape and size, the dendritic processes were not entirely perfect, the nuclei were central and the cells were normal in number. This was an excellent case for the study of tertiary degeneration, as the lesion occurred at the third year of life, and the cell-bodies of Clarke's column had disappeared as a result of retrograde atrophy, but tertiary degeneration if it existed had not caused much alteration.

I have examined by the carmine stain a case of spinal hemiplegia from a lesion in the cervical region which had existed for a number of years. The cell-bodies in the lumbar region on the side of the degenerated crossed pyramidal tract were numerous, the nuclei were central, but the dendritic processes were possibly not perfectly developed.

Of course the cells in both these cases might have shown some alteration if Nissl's method had been employed, but I chose two cases of lesion of the lateral columns in the cervical region to show that atrophy of the cell-bodies of the anterior horns detectable by carmine does not occur to any extent from degeneration of the lateral columns. Possibly a lesion developing even earlier than the third year of life might give different results, or I might have obtained chromatolysis by Nissl's method in these cases. I have recently obtained a spinal cord which was greatly compressed in the upper cervical region.

by an intradural tumor. The patient had been paralyzed in all the extremities. Many of the cell-bodies in the anterior horns of the lower cervical region are much diseased and are in a condition of central chromatolysis and vacuolation with displacements of the nucleus (Fig. IV.). In the lumbar region the altered cell-bodies are much less numerous. This is possibly tertiary degeneration, but some of it may be "reaction at distance" from the injury of the axones of cells located lower in the cord.

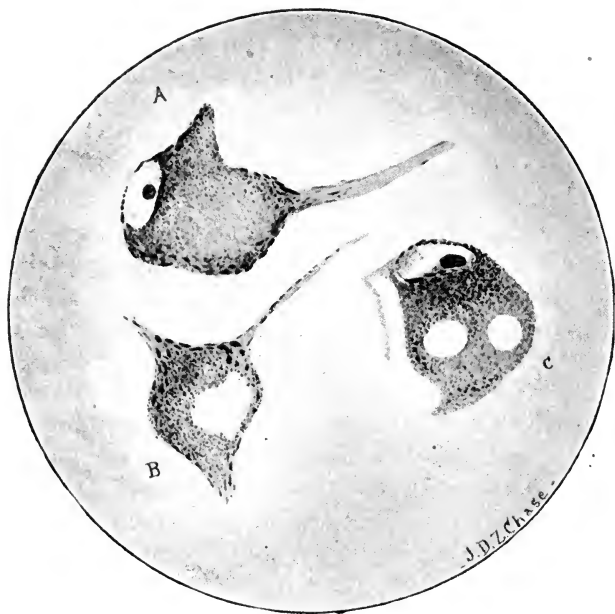


Fig. IV. Cell-bodies of the anterior horns in a case of tumor of the upper cervical region. Many of the cells show central chromatolysis and vacuolation. Cell-body A is from the lumbar region, B and C from the lower cervical. The cellular changes may be "reaction at distance" from injury of the axone, or may be tertiary degeneration of the peripheral motor neurone.

I am at present studying a case of very great value—one of cerebral diplegia from an unilateral lesion. The history is imperfect, but it is positively asserted by thoroughly reliable persons that four and a half years before the boy's death he had been able to walk, but his gait was probably not normal.



He soon lost this power after he came under observation, and his limbs became helpless and contracted. I found intense internal hydrocephalus limited to the right hemisphere, and this hemisphere was a thin-walled sac. The motor fibers were greatly injured and the left crossed pyramidal tract was sclerotic, and yet the cell-bodies in the anterior horns of the cervical and lumbar regions seemed to be normal in number, and little change could be detected by Nissl's method. The boy was fourteen years old when he died. Surely this was a very suitable case for testing tertiary degeneration in the peripheral motor neurones.

Schaffer,<sup>27</sup> however, has observed chromatolysis of the spinal motor cell-bodies in recent hemiplegia, and this chromatolysis was especially noticeable in the postero-lateral group. Quite different are the results obtained by Marinesco.<sup>28</sup> In sixteen cases of hemiplegia with degeneration of the pyramidal tract, that he examined, he found alteration of the cell-bodies in the anterior horns of the cord only in three, and in two of these cases the altered cell-bodies were not numerous and might easily have escaped detection. In the case in which the changes were distinct they were like those seen when an axone is cut, and were suggestive of neuritis.

We should be exceedingly cautious in accepting diminution in the size of one anterior horn as a result of degeneration of the pyramidal tract. In the case of malaria of the central nervous system, the report of which I present at this meeting, a slight sclerosis existed in one crossed pyramidal tract and the anterior horn on that side throughout the cord was distinctly smaller than the horn of the other side. The number of the cell-bodies was about the same on the two sides. The sclerosis, was so slight and the symptoms it caused so transient, that I look upon the diminution in size of the anterior horn occurring with the slight sclerosis of the motor tract merely as a coincidence.

Berger<sup>29</sup> has found very distinct degeneration of the cell-

<sup>27</sup>Schaffer, *Monatsschrift für Psychiatrie und Neurologie*, Vol. 2, No. 1.

<sup>28</sup>Marinesco, *Semaine méd.*, 1898, p. 465.

<sup>29</sup>Berger, *Monatsschrift für Psychiatrie und Neurologie*, Vol. 3, p. 1.

bodies of the anterior horns in paretic dementia. This degeneration might have been regarded as due to toxic causes or to degeneration of the pyramidal tracts. The experiments Berger performed are of much interest in the light of Warrington's<sup>30</sup> positive findings. Berger was unable to produce in the dog degeneration of the contralateral anterior horn cell-bodies by destruction of the motor cortex, although the pyramidal tract in the cord was degenerated. In another dog he destroyed the cortical motor area and divided the contralateral posterior roots and allowed the animal to live four weeks. The cell-bodies of the anterior horns were not degenerated. In a cat in which the pyramidal tract of one side had been destroyed for two years no cellular changes in the corresponding anterior horn were found. Dr. McCarthy, at my request, has kindly cut for me posterior roots in three cats. One cat was allowed to live seven days, another nineteen days, and the third twenty days. The examination of the spinal cord from these animals has given results similar to those obtained by Berger. I find no distinct alterations of the cells of the anterior horns resulting from division of the posterior roots.

The results of these experiments cannot be applied equally well to man. The dog and cat seldom use one limb separately, and probably all four limbs are innervated from both sides of the brain more nearly equally than they are in man. To cut off the stimulation from the cortex in the dog it would be necessary to destroy both motor tracts, and possibly even then this would not be sufficient on account of the existence of "extra-pyramidal" tracts.

I have re-examined the sections in the case of syringomyelia reported by Dr. Dercum and myself<sup>31</sup> before this Association four years ago. The posterior horn of the right side was destroyed from the first thoracic segment throughout the cervical region, and in the lower part of the cervical region the crossed pyramidal tract on the side of the destroyed posterior horn exhibited distinct retrograde atrophy. The lesions had existed for a number of years and the cells of the right

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<sup>30</sup>Warrington. *Journal of Physiology*, Vol. XXIII.

<sup>31</sup>Dercum and Spiller, *The American Journal of the Med. Sciences*, 1896.

anterior horn had been deprived of all stimulation through the right posterior cervical roots, and to some extent of the stimulation from the brain through the right motor tract of the cord, on account of the retrograde atrophy of the right crossed pyramidal tract. There may possibly be a slight decrease in the number of cell-bodies in the right anterior horn, but this decrease is questionable and the cell-bodies present appear to be normal by the carmine stain, and have well-developed dendritic processes and central nuclei. The case does not give any positive evidence of degeneration of the cell-bodies of the anterior horn from lesions of the posterior roots.

Degeneration of the peripheral motor neurone certainly may exist at least many years without degeneration of the central motor neurone. I refer in proof of this statement to the interesting and much cited case reported by Senator. The symptoms of amyotrophic lateral sclerosis existed for about five years, and Senator<sup>32</sup> found a pronounced simple, non-inflammatory atrophy of the cell-bodies in the anterior horns of the cervical and thoracic regions. The anterior roots were not distinctly diseased. He says positively that lateral sclerosis was *entirely* wanting.

Dejerine's<sup>33</sup> cases are further proof in this respect. In two cases of progressive spinal muscular atrophy the peripheral motor neurones were intensely diseased, but absolutely no degeneration of the pyramidal tracts was detectable. One case lasted eighteen years and the other ten years. These specimens Dr. Dejerine has permitted me to study. Similar cases have been reported by Dreschfeld, Oppenheim, Nonne, Dutil and Charcot.

I have never seen involvement of the lemniscus as a result of degeneration of the posterior columns. Two cases in literature in which this was reported occur to me. One was a case reported by Schaffer<sup>34</sup> and is of most difficult interpretation; the other is by Rossolimo.<sup>35</sup> In the latter the portion of

<sup>32</sup>Senator, Deutsche med. Wochenschrift, No. 20, 1894, p. 433.

<sup>33</sup>Dejerine, Comptes rendus de la Soc. de Biologie, 1895, p. 183.

<sup>34</sup>Schaffer, Archiv für mik. anatomie, Vol. XLIII.

<sup>35</sup>Rossolimo, Archiv für Psychiatrie, Vol. XXI.

medulla oblongata containing the nuclei of the posterior columns was not examined, and these nuclei may have been altered independently of the degeneration of the posterior columns. I have traced intense degeneration by Marchi's method from the sacral cord to the nuclei of the posterior columns, but never beyond.

Schaffer<sup>36</sup> thinks that the degeneration of the direct cerebellar tract in tabes is an example of the influence of peripheral sensory neurones on central sensory neurones. I know such cases have been reported, but I have never been fortunate enough to observe any, although I have examined many cords from tabetic persons. I think, therefore, that this degeneration must be rare. Schaffer, however, can give no example of the influence of a central sensory neurone upon a peripheral sensory neurone. He has found central chromatolysis and displacement of the nuclei in the cell-bodies of the anterior horns in tabes and he attributes these cellular changes to the degeneration of the sensory roots. The changes, however, may be due to the peripheral neuritis occurring in tabes. I also have seen this chromatolysis of the cells of the anterior horns in tabes.

A statement made by van Gehuchten is of no value as evidence of tertiary degeneration. Van Gehuchten<sup>37</sup> observed chromatolysis of the terminal nucleus of the acoustic nerve after division of the nerve, and reported this finding at the meeting of the Congress in Moscow. He explained this chromatolysis by the theory of Marinesco and regarded it as the result of injury of the first neurone and of loss of irritation normally conveyed by this neurone. Further researches convinced him that his technique was at fault. The view he now holds is that division or injury of a peripheral sensory nerve does not cause early changes in the cell-bodies forming the terminal nucleus of this nerve. He has not, therefore, seen chromatolysis in the terminal nucleus as a result of injury to the peripheral sensory neurone.

I know of no positive evidence that disease of a sensory neurone ever results from disease of a motor neurone.

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<sup>36</sup>Schaffer, *Monatsschrift für Psychiatrie und Neurologie*, Vol. 3, p. 64.

<sup>37</sup>Van Gehuchten, *Journal de Neurologie*, Nec. 20, 1898, p. 502.

I am, of course, well aware of Marinesco's<sup>38</sup> views. He teaches that every disturbance of function in a neurone affects the function of the neurone with which it is in connection. Lesion of a sensory neurone causes in course of time alteration of a second sensory neurone of a different series, possibly even of a sensory neurone of a third series. Lesion of the cortical substance causes similar neuronic atrophies, but in an opposite direction. This theory sounds so plausible that we cannot reject it, but my researches cause me to believe that the influence of one neurone upon another has been to some extent exaggerated.

I would not give the impression that tertiary atrophy—I use the word atrophy and not degeneration—does not occur. I believe that when the function of a neurone is destroyed early in life, even though the neurone is not directly injured as, for example, by division of its axone, this neurone whose function has been arrested develops very imperfectly or possibly even atrophies. The diminution in the size of the lateral column following amputation of a limb is regarded as an evidence of this, and I have seen slight diminution of the antero-lateral column following early amputation. Tertiary degeneration in the adult—possibly it is never more than atrophy—I believe to be rare, especially in the motor system. Tertiary atrophy is possibly more common in the sensory system or in the visual tract, and possibly the explanation of this difference lies in the fact that the peripheral motor neurones are under the stimulation of both the pyramidal tracts and of the sensory roots.

The fact that two neurones in connection with one another do not degenerate to the same degree and with the same rapidity from one lesion is evidence that there is some difference in structure at the point where they come together, and that the essential elements of one neurone are not the same essential elements of another neurone.

That degeneration may occasionally pass from one neurone to another is no proof that neurones do not exist. It is certainly not common for systemic diseases to spread irregularly to neighboring areas. The views of Held, Apáthy, Bethe and Nissl may force us to modify our conceptions of the neurone, but the evidence offered by pathology of the existence of neurones, *i. e.*, of structures having a functional individuality, will have to be harmonized with the results of anatomical studies.

<sup>38</sup>Marinesco, *Presse méd.*, 1898, p. 201.

(c) HOW DOES THE NEURONE DOCTRINE AFFECT THE  
CONCEPTION OF NERVOUS DISEASE?\*

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The introduction of the neurone theory promised to bring about a general revolution in the conception of nervous disease. With a single swoop the mysteries and doubts of years were to be cleared away. Granted that the structure and function of the nervous system can be interpreted much more readily in the light of this doctrine, it still remains a question whether it has been a great assistance to us in determining the causation and development of the various diseases of the nervous system. The researches of Golgi, Ramón y Cajal, van Gehuchten, Edinger, Koelliker, Waldeyer, and the excellent work of Barker,<sup>1</sup> have kept us all in touch with every stage of progress in the development of the neurone theory. It was a matter of surprise to me, however, that, in spite of the interest taken in the theory, I found myself making but little reference to it in clinical lectures. The theory did not seem to add much to the teachings of former years in interpreting the various forms of brain and spinal cord disease, and other teachers of neurology, men whose opinion I value highly, have confessed to similar experiences. It is also to be noted that authors of standard works, with the possible exception of Leube and Struempell, have had little to say about the theory in its bearing upon disease, although many of them refer to the theory in an introductory chapter. With every disposition on my part to turn the new theory to fullest account, it seemed well to attempt an impartial answer to the question of the utility of this new theory in clinical teaching.

To the clinician the following points in the neurone doctrine have a special significance: It does away with the somewhat ar-

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\*Read at the twenty-sixth annual meeting of the American Neurological Association, May 1, 2 and 3, 1900.

<sup>1</sup>The general literature of the subject may be found in Barker's work, "The Nervous System and Its Constituent Neurones," N. Y., 1899; and in the monograph by D. I. Wolfstein, of Cincinnati, "The Neurone Theory as Revealed to Brain and Nerve Diseases," Providence, 1899.

bitrary division of cerebral, spinal and peripheral nerve disease; and a decided gain this is, for morbid processes do not always respect artificial lines; parts functionally related are apt to be simultaneously affected. It has been shown that the nervous system is composed of a series of contiguous, not continuous units; that in this nerve unit, composed of the cell-body, with its dendrites, the nerve fiber or neuraxone and the terminal tufts, the cell-body exercises an important trophic influence over the entire neurone; furthermore, that the nerve force, to speak in general terms, could travel from the cell-body to the periphery, and from the periphery to the cell-body. In the ante-neurone days the same nutritive or trophic influence over the nerve fiber was ascribed to the "cell" proper, with this difference, that we did not claim as intimate a relation between the distant nerve fiber and the ganglion cell, with which it was connected, and from which it was supposed to spring. As far as the performance of function is concerned, it matters little whether there is actual contact between terminal fibrils, or mere contiguity; the "nerve force" must in the end pass, and pass somehow from one nerve unit to the next.

With the promulgation of the neurone theory, and the division into motor and sensory neurones the laws of Waller and of Türeck were not in anywise affected, but admitted of an easier interpretation. Degeneration would set in in any part of the neurone severed from the cell-body, and in case of such severance the distal end would be the first to suffer. Goldscheider<sup>2</sup> is cautious enough to state that this does not preclude the possibility that a part of the neurone distant from the cell-body may take its nutrition from its immediate environment, but he thinks that such assimilation cannot be properly effected, except under the influence of the cell-body. Great as this influence of the cell-body may be over every part of the neurone, the upward degeneration which sets in after amputation of the nerve fiber proves that the health of the cell-body depends also upon the health of the distal parts. This reflection has led Marinesco<sup>3</sup> to develop the doctrine that, if the normal function of the cell-body is to

<sup>2</sup>Berliner klin. Wochenschr. 1894, Nos. 19 and 20.

<sup>3</sup>Neurolog. Centralblatt 1892. P. 463 *et seq.*

be preserved, it must receive normal peripheral stimuli. The life of the cell-body depends upon the life of its terminal parts—in other words, the cell-body and nerve fiber are interdependent; both must remain normal if the nerve unit is to perform its normal function. The case of the cell-body—to speak in legal terms—has been overstated; for the nerve fiber it has been insufficiently presented. The investigations of Ballet and Dutil, which Barker quotes, prove that changes take place in the ventral horns in polyneuritis. It will not be amiss to present to our minds, in the case of the direct motor neurone, the great distance that intervenes between the distal end of the nerve fiber in the extremity and the cell-body in the spinal cord. We must take into account the importance to this distant part of a normal blood supply (to mention only a single condition) for the preservation of a nerve fiber, and it is evident that there are very important conditions, other than those inherent in the cell-body, upon which the health of the entire neurone must depend. We have done the peripheral fiber an injustice by making it altogether subordinate and inferior to the cell-body; it is at least co-ordinate with it in the same sense that in an electric system the wire is of no account unless the home station is in good working order; but the home station is also unable to perform its work if the wires are broken down, and they may and do break down quite as readily as the central station itself. The distal parts of the neurone—the wires—are subject to disturbances of their own. This has been shown in the case of the effect of the various toxic agents, for these seem to exert their influence chiefly upon the distal parts of the neurone, and possibly in secondary fashion only upon the cell-body. The farther away from the cell-body the diseased fiber lies, the greater the damage; possibly, also, the greater the difficulty of repair. It would be a natural and logical consequence to expect alterations in the cell-body whenever the peripheral parts of the neurone is affected by disease, by poisoning, or by injuries of any sort. It may be claimed that former methods were insufficient to prove that such changes actually occurred in the ganglion cell, and that it will require careful examination by the most recent staining methods to prove or disprove the intimate relation be-



tween the fiber and the home station. To uphold the full dignity of the cell-body and to account for the fact that its structure, as far as at present made out, does not seem altered for a long period of time after the distal parts have been affected, Erb and others have formulated the theory that the cell may be altered in function, and thus exert a perverse and insufficient influence over the distal parts, without exhibiting any great change in its structure. To such straits are we put in upholding the paramount influence of the cell proper. Attractive as this theory is, there is danger lurking in it, for we are piling theory upon theory, without having sufficient evidence, or without even the hope of being able to give substantial proof of the correctness of this additional doctrine. It will surely be safer, in view of all these perplexing theories and explanations, to hold to the statement that the cell-body and nerve fiber are functionally co-ordinate and equally important parts of the entire neurone system.

This question of the trophic influence of the cell-body had to be discussed before we could attempt to appreciate the value of the neurone in its relation to disease of the nervous system. The doctrine helps us, of course, very little in our understanding of the acute and inflammatory processes, for such processes do not respect the finer structure of the brain or cord, and are generally so destructive that it would matter little whether the nervous system were made up of contiguous or continuous units. Allusion was made above to toxic diseases, and the neurone doctrine has been of the greatest possible assistance in reconciling the different findings that have been reported by various authors in regard to the effect of poisons upon the peripheral nerves and the spinal cord. It was difficult in former days to account for the claim put forth by many that in lead poisoning the peripheral nerves were diseased, while the experiments upon animals by Stieglitz and the post-mortem findings in the human body showed that in extreme cases, or at least in some instances, the cells of the spinal cord were affected as well. It simplifies matters very much to be able to claim that lead and other poisons exert their chief influence over the direct motor neurone, and that it matters little whether in a given case the central or distal part of the neurone may be in-

volved. The doctrine also helps us to account for the occurrence of polyneuritis and poliomyelitis in those rare cases that have been reported by Gowers, and we must suppose that organic as well as inorganic poisonous products may affect the various parts of the peripheral neurone; in the interpretation of such conditions the doctrine has been distinctly helpful.

Much has been claimed for the aid which the neurone doctrine offers in the interpretation of the degenerative diseases of the central nervous system, whether such degeneration affect primarily the gray or the white matter. *Tabes dorsalis* is to be accepted as a disease of the direct sensory neurone; whatever the nature of the poison circulating in the body may be, the sensory fiber is the part primarily affected. Even if the view could be maintained that the disease began in the spinal ganglion cell, this ganglion cell would not be diseased unless there was something wrong about the peripheral stimuli conveyed to it. But is there any evidence that the disease does actually begin in the most distal parts of the neurone? Wolfstein, who has written an excellent summary of the entire question, refers to the fact that Batten has shown that in *tabes* the muscle spindle presents the distinct evidence of disease. We have here a peripheral organ of sense, as this author says, which transmits external impressions to the peripheral sensory fiber, which is seen to undergo degeneration. "Armed with the hypothesis of Bechterew of the vital influence of the sensory end apparatus upon the character of the afferent impressions and with the theory of Marinesco regarding the necessity for the continuance of the function, in order that the nutrition and the power of the trophic center be maintained, we can readily understand the changes which follow the degeneration of the sensory ending." Plausible as this explanation is, it must be claimed that such muscle spindle changes have not been established in a sufficient number of cases to warrant us to utilize them in the attempt to explain the origin of the morbid changes of *tabes*; and, without being facetious, we might supplement Wolfstein's remarks by saying that if we are armed with hypotheses enough, we can explain almost anything and everything. If Fraenkel is right in maintaining that even the motor

palsies of tabes are dependent upon changes in the deep sensibility, such palsies would not militate against the neurone theory of the disease, and possibly Batten's findings may also explain the occasional occurrence of marked muscular atrophies in tabes. After all has been said that can be said in favor of this newer doctrine, we now speak of tabes as a disease of the direct sensory neurone, whereas in former days, we considered it a disease primarily affecting the sensory nerve systems. In view of the probable and frequent syphilitic origin of the disease, it is of course helpful to know that the products of syphilitic poisoning affect chiefly the direct sensory neurone; other poisons having a predilection for the motor neurones.

A consideration of the neurone doctrine has also led to a better understanding of the spinal form of progressive muscular atrophy, and of its relation to progressive bulbar palsy and to amyotrophic lateral sclerosis. The first two forms evidently represent primary degenerations of the direct motor neurone, either in the spinal cord or in the lower portion of the brain axis. Of course, we are still at a loss to know why such degeneration should set in in the few individuals affected by the disease, but we accept such degeneration as a fact. Amyotrophic lateral sclerosis would in the light of this theory represent the same primary degeneration affecting the cortical cells of the indirect motor neurone, as well as the direct motor neurone; or, as Kahler and Moebius put it, amyotrophic lateral sclerosis represents a primary degeneration of the various portions of the cortico-muscular tract. But in this disease in the direct motor neurone the cell-body is the part primarily affected; in the indirect motor neurone the distal part is the part first diseased, and the attempt to show that these distal parts are dependent upon functional, if not structural, changes in the far-distant pyramidal cell-body has not been entirely satisfactory. To be sure, such changes have possibly not been established because the necessary investigations have not yet been made. But here again it will not do to pile theory upon theory, until concrete findings shall have been reported which tend to uphold this view. It is not an unnatural thought to suppose that in a form of cord disease of one neurone, it may in some instances spread to the distal part of a neighboring

neurone, and it seems much more plausible to think of the morbid influence exerted by a degenerated or degenerating neurone upon neighboring fibers than to suppose that such degeneration in the direct motor neurone is due to problematic affections of the distant pyramidal cell. I shall be inclined to the opinion that even if, in some instances, changes in the pyramidal cells were found, that these would be entirely secondary to the changes in the distal parts.

It would be natural also to expect further light from this doctrine upon the relation of the various forms of progressive muscular atrophy to the Aran-Duchenne type. It is surprising to know that no author has as yet referred to the controversial findings in the case of the peroneal form of progressive muscular atrophy. Hoffmann, as is well-known, maintains that it is due to a progressive neuritis—in other words, that it is neural in origin; whereas, equally reliable investigators have shown that in some instances the spinal cord has been involved. It is always difficult to ascertain whether such involvement is primary or secondary; but the conflicting views can be reconciled if we regard this form of progressive muscular atrophy as a degeneration of the direct motor neurone; the degeneration in some instances affecting the distal parts and in some the central part of the neurone. Some doubt has of late been cast upon the radical differences which were supposed to exist between the amyotrophies and the progressive muscular dystrophies. It is possible, if not probable, that the muscular fiber is subject to primary degeneration, without any reference to the terminal nerve plate, but it would be a grateful task to examine the peripheral nerve endings in the earlier stages of the progressive dystrophies, to determine whether or not these are altered. Such changes as I found in the peripheral nerve in a typical progressive muscular dystrophy might well be secondary to a primary degeneration of the muscle, for any marked change in the muscular tissue by removing the normal peripheral stimulus should in the course of time affect the peripheral nerve endings. On this special point I am in hopes of shedding some light by an examination of a case of progressive muscular dystrophy now under investigation. The neurone doctrine, if it does not clear up a number of the ques-

tions still under dispute regarding muscular dystrophies, will at least give additional interest to the investigation of these diseases, which seemed only a short time ago to have been thoroughly studied and almost exhausted.

The combined sclerosis of the cord which have been so difficult to explain may also admit of somewhat readier interpretation, if we choose to accept the view at which I hinted that the disease of one neurone may affect the neighboring distal parts of another neurone. The older conception of the systemic diseases of the central nervous system will, no doubt, have to yield to the more modern doctrine that disease affects parts functionally if not structurally related. It will also tend gradually to minimize the importance of the older anatomical divisions of nervous disease into diseases of the brain, of the spinal cord and of the peripheral nerves, and no doubt the general interpretation of disease will be materially helped if we substitute, as far as possible, physiological for the older and anatomical sub-divisions. It is not necessary to enter upon a discussion of the aid rendered by the neurone doctrine in the interpretation of mental diseases. The attempt has been made to apply the doctrine to the elucidation of general paresis. To say that the associative, motor, and sensory nerves are involved is to hold that every part of the central nervous system is more or less diseased; it would be of some interest, and would bring out in the strongest light the relation of general paresis to tabes, if it could some day be shown that in general paresis also, the sensory neurones are primarily affected. It is altogether premature to attempt an explanation of other forms of mental derangement in the light of the new theory. I cannot sympathize with a recent author (Wolfstein) who believes that the neurone doctrine "has reconciled the physical and the metaphysical, it has bridged over the chasm between the material physiologist and the metaphysical physiologist, and joined their hands in harmony."

Nor can I find sufficient reason for the present to apply the neurone doctrine to the interpretation of functional diseases and functional conditions. To speak of irritable sensory neurones is just about as vague as to refer to spinal irritability, as we did in former days. A substitution of words does not

imply an increase in our own concepts. The defense of the retraction theory I must leave to one of its chief apostles, who is fortunately in our midst.

Let me conclude by saying that the neurone theory is of distinct assistance in the interpretation of toxic diseases, affecting at one and the same time the peripheral and central nervous systems, while in some instances involving only one or the other, and not both parts; it has shed some light upon the perplexing questions of the relations of the various forms of progressive muscular atrophy, and upon the early morbid changes in tabes. It may be of some further help in our understanding of the combined scleroses. More than this can scarcely be claimed for it; if it shall have accomplished all of this, and shall be able to stand the test of further sane criticism, it will be entitled to the consideration it has already received.

100 UN CAS DE TORTICOLLIS MENTALE (A Case of Mental Torticollis. E. Nougès et J. Sirol (Nouvelle Icon. de la Salpêtrière, Nov. et Dec., 1899).

Brissaud first called attention to this form of spasmodic tic. The condition is not very rare; the case is as follows: A gardener, thirty years old. Father and grandfather had stuttering speech. The patient began to stammer at the age of eight years; this condition increased in intensity up to twenty years, when it diminished somewhat, manifesting itself only at times of emotional excitement. In the month of September, 1898, without any apparent cause he felt his head drawn to the right side; shortly after this, to the left side. On attempting to restrain this movement, a spasmodic contraction of the sternocleido-mastoid muscle was produced; after a few contractions, the head was in its normal position. These movements increased in variety and frequency, the trapezoid muscle taking part in them. He learned to stop them after a time by pressure of the fingers on the opposite side of the chin, nose, etc. There is no doubt that the condition in this case was exclusively psychical. Bempaire has said that in all cases of mental torticollis, evidences of degeneracy are to be found. In this case there was present hereditary condition of stuttering, which points in the same direction. The author concludes that mental torticollis is a spasmodic tic, and all tics of a mental origin develop often, if not always, in degenerates. SCHWAB.

## Periscope.

### CLINICAL NEUROLOGY.

101 HEILUNG ACUTER GEISTESSTÖRUNG NACH EXTIRPATION EINER HIRNGESCHWULST (Cure of Acute Mental Disturbance after Extirpation of a Brain Tumor). Bayerthal (Münchener medicinische Wochenschrift, 1899, No. 46, S. 1537).

A man of twenty-nine, having had in his eighteenth year cervical adenitis, but otherwise having been healthy, began early in 1897 to suffer from headache and vertigo. On April 1 he noticed a weakness in the left foot, and ten days later had an epileptiform attack, beginning with twitching in the toes of the left foot. Similar attacks recurred from time to time, and the patient's condition grew gradually worse.

At the time of coming under the author's care in December, 1897, there was paresis of the left leg and weakness of the left arm, with exaggerated reflexes in the leg. He also suffered from paresthesia in the left lower extremity, and had occasional attacks of precordial oppression. He was treated by bromide and iodide of potassium, until June, 1898, but with no improvement. At this time there appeared clonic contractions of the muscles of the left arm, which much distressed the patient. In September, 1898, he developed visual hallucinations with delusions of persecution, and began to threaten his surroundings. On account of the leg paralysis, of the signal symptom noticed at the beginning of the Jacksonian attacks, and of localized tenderness on percussion in the right parietal region near the middle line, a tumor situated in the right paracentral lobule was diagnosed, and an operation was decided upon. Under morphia-chloroform narcosis, a skin and bone flap was raised from over the upper end of the central convolutions, but the operation had to be interrupted, on account of threatened heart failure. Two days later it was completed under morphia alone, without difficulty, and apparently without pain to the patient. Upon opening the dura, the brain bulged slightly, and appeared entirely normal. Upon careful inspection, however, there was seen at a point very near the median line a slight injection of the pia, in the middle of which there were a few tiny nodules. Careful probing at this point disclosed the presence, at about  $2\frac{1}{2}$  mm. beneath the surface, of a hard mass. A finger introduced was able to shell out a tumor, which arose from the median surface of the hemisphere, and sent a process about 2 cm. long, and 3 cm. thick, forward and outward. The bleeding was inconsiderable and was easily controlled. The tumor, which was about the size of a walnut, proved to be a solitary tubercle. The patient made a good recovery. His mental condition became normal at once; only on one occasion was there recurrence of hallucinations, and they were then very fleeting. There was some gain in power in the left leg, and the twitching in the left arm gradually disappeared.

Two months after the operation the patient was discharged from the hospital. Mentally he was quite normal, except for slight irritability. There was still left hemiparesis, the leg much more affected than the arm, being dragged somewhat in walking. There was paresthesia in the affected limb. One year after the operation, the patient still remained in good mental condition; there was some

increase in power on the paralyzed side, but the Jacksonian attacks had recurred, the patient having had four in spite of bromide treatment. These attacks always began in the left leg. ALLEN.

102 CASO DI TETANO TRAUMATICO CURATO COLL'ANTITOSSINA TIZZONI-GUARIGIONE. (A Case of Traumatic Tetanus Treated by Tizzoni's Antitoxin—Recovery). Virgilio Barachini. (Gazz. degli Ospedali et delle Cliniche, Jan. 7, 1900. No. 3, p. 20).

The author relates the case of a previously sound boy of thirteen years of age. Seventeen days before the onset of his nervous symptoms, he had, while playing barefooted with some companions, accidentally thrust the prong of a sharp pitch-fork into the sole of the right foot, producing a wound 2 cm. long and  $2\frac{1}{2}$  deep. He made no attempt to cleanse the wound, and kept about his play during the entire day. Four days later Barachini was called to see him because the wounded foot, which had as yet received no medical treatment, had become red and swollen, and the boy feverish. B. carefully cleansed the wound and applied antiseptic dressings. Under this treatment suppuration ceased, the temperature dropped, and a complete cessation of all symptoms in a short time was confidently looked forward to. Within a week, however, the boy's parents noticed an unusual inappetence on his part and also that he had become restless and no longer cared for play.

On the evening of the 17th day after the accident, the boy, on attempting to withdraw from the supper table felt his entire right leg grow stiff, and immediately after his whole body was shaken by a convulsion so painful that he fell to the ground. In his fall he struck his head violently and remained unconscious for some time. On recovering his senses the pain was found to have disappeared, but the muscles of the trunk and lower extremities were so rigid that he had to be carried to bed. During the night he experienced general malaise and an intense pain which radiated from the injured foot over the entire right leg and invaded the left to a less degree; he also felt rigid all over, but particularly in the jaws, neck, back and lower extremities, the rigidity being most marked during an access of pain. To this was added a painful sense of constriction in the throat, with dysphagia. The next morning Barachini found the boy in bed and presenting the tetanic facies, with risus sardonicus well marked. Trismus was so pronounced that it was only with the greatest difficulty that the handle of a teaspoon could be introduced between the teeth, and efforts at swallowing even small quantities of liquid increased the spasm of the face and neck muscles. The wounded foot showed only a small dirty-gray suppurating area.

Barachini at once administered a subcutaneous injection, in the axillary region of the left chest, of 25 cm. of Tizzoni's anti-tetanic serum, equivalent to 2,500,000 immunizing units, and prescribed a small amount of bromide and chloral. During the night the patient slept somewhat and had only three or four convulsive seizures; he succeeded, also, in swallowing liquids fairly easily. The next day the trismus was increased, but temperature, pulse and respirations had fallen. A second injection of an equal amount of the serum was given on the opposite side of the chest, but without result, and in the evening 1 ctg. of morphine was injected. The following day there was noticeable amelioration of all symptoms, with a further drop in pulse and temperature. No further use of the serum was made, but the bromide and chloral were given by the mouth on two occasions, and the bowels were moved with calomel and syrup of buckthorn. Six days after the onset of the tetanic symptoms a



very marked eruption of urticaria was noticed over the sites of the serum injections, and this eruption gave rise to so much agitation on the part of the patient that the tetanic phenomena returned in a measure, but were controlled by a clyster containing 2 gm. of chloral. Three days later the boy left his bed completely well. Total duration of symptoms nine days.

In discussing the case the author thinks the effect of the bromide and chloral to have been only minimal, and gives the credit for the cure entirely to the serum. He states, further, that his case is the sixth which has been treated with the serum at Pisa, and that the outcome in all has been successful.

J. W. COURTNEY.

### 103 RECIPROCAL INFLUENCE OF EPILEPSY AND PREGNANCY.

Chambrelet (*J'l de med. de Bordeaux*, Nov. 5, 1899), states that in 1884 Beraud published a thesis (Paris), in which he investigated this entire subject to date. He found notes in literature of some thirty cases of epileptic women who bore children. In one-half of this number the epileptic condition was benefited by pregnancy, the seizures either being absent altogether or much lessened. In seven cases the state of pregnancy exerted no influence upon the course of the epilepsy, while in the remaining eight cases the latter was rendered decidedly worse. Since the date of Beraud's thesis a number of cases have been reported, the most important series being that of Pinard, who noted nine instances of improvement (including four of cessation) and two cases of negative result, but none which was worse during pregnancy.

The combined statistics of Beraud and Pinard show improved, 24; stationary, 9; aggravated, 8. Chambrelet reports two cases, one being much better (cessation), and one worse through pregnancy. Charpentier records a case in which, during her first pregnancy, the woman became much worse, and during her second pregnancy she died of status epilepticus. Chambrelet regards it as impossible to give any reason as to why one case should improve and another grow worse on account of pregnancy.

Epilepsy has no influence whatever upon the mother, and there is no record of an epileptic mother having aborted as a result of convulsions during pregnancy. Even the author's patient, who died of status after extreme convulsions, did not abort. In the case of the effect of epilepsy upon the new born child, statistics are wanting, but it is evident that a very large proportion of children suffer from convulsions a few hours after birth, while of this number many perish. Possibly some relationship might be shown between cases which become worse from pregnancy and those who bear children who develop convulsions.

CLARK.

### 104 EIN FALL VON TRAUMATISCHER PERIODISCHER LÄHMUNG (A Case of Traumatic Periodic Paralysis). J. Donath (*Wiener klin. Woch.*, 13, 1900, p. 36).

No history of periodic paralysis in any member of the family, except in one case, was obtained. A woman of twenty-five years met with a severe accident, and three days later became suddenly paralyzed in her entire body. She had never previously had similar attacks. The attacks lasted from half an hour to eight days and were numerous, and when they were severe even the head could not be moved. Electrical reactions were lost during the paralysis. Occasionally the attacks were abortive. The occurrence of the attacks of paralysis for the first time after a trauma was remarkable in this case.

SPILLER.

105 NOTE SUR DEUX TICS DU PIED (Note on Two Cases of Tic of the Foot). F. Raymond and Pierre Janet (*Nouvelle Incon. de la Salpêtrière*, Sept., Oct., 1899).

These two cases illustrate conditions of tic due to "*automatisme psychologique*," and are described to show that various parts of the body may be attacked by tics of such a nature. They both show a tic which is of psychical origin.

*Case 1.*—A woman, aged thirty-seven years, complained chiefly of an inability to move the left foot. This foot was held in a varus position, the big toe being placed in forced extension, while the three middle toes, and especially the little toe, were separated from each other, being extended outward. This position of the toes caused great pain when the patient attempted to walk. At first sight this might be considered as a contracture, possibly of hysterical origin, but when the patient was at rest contraction disappeared. Examination of the leg showed nothing abnormal in regard to sensation or motion, so the diagnosis of a tic, analogous to a spasmodic torticollis, was made. The origin of the condition was purely psychical. Patient had syphilis seventeen years ago. Seven years ago she was treated for some ocular condition with inunctions of mercury. At the same time she suffered from a corn on the left foot. Thinking that the mercury would be good for that also she applied it to the corn in question. The application was very painful and frightened the patient very much. The next day she observed that the foot was cramped. The present condition dated from that time.

The second case is that of a man who walked with great difficulty on account of a spasmodic condition of the toes. The psychical origin in the second case was less clear; it dated from the wearing of a badly fitting shoe. The diagnosis of hysterical contraction was excluded on the same grounds as in the first case.

Treatment in the two cases was identical. An effort was made to convince the patients of the nature of their trouble. After they were aware that the whole thing was merely a habit, exercise treatment was instituted, during which their attention was directed to the movements of the spasmodic muscles. In the first case suggestion in addition was tried and was easily produced. In the second case, improvement was more tedious, but the man was taught to walk after some time. These two cases by their evolution and treatment confirm the idea that the mechanism of the condition is purely a psycho-physiological one.

SCHWAB.

106 ANALGESIA OF THE ULNAR NERVE IN EPILEPTICS.

Lannois and Carrier (*Revue de med.*, 1899, Nov.) state that Bier-nacki first made this discovery in connection with tabes, and Cramer afterwards studied it in paralytics. Hildenberg soon after investigated the matter in regard to epilepsy and found the symptom in 75 per cent. of 53 patients. Many other investigators, however, have failed to find this symptom. The present authors therefore sought to decide the matter. One hundred and three epileptics were tested with regard to sensitiveness on pressure over the ulnar nerve at the elbow. In 50 males sensibility was normal in 27 cases, depressed in 11, absent in both sides in 5 cases, and on one side in 4. Of the 80 females sensibility was normal in 40, depressed on both sides in 11, absent in 14 (both sides), absent on one side in 10. It is evident, therefore, that this symptom is by no means characteristic of epilepsy, and when found merely signifies nervous exhaustion.

CLARK.

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**Original Articles.**

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A CASE OF ADIPOSIS DOLOROSA, WITH NECROPSY.\*

By CHARLES W. BURR, M.D.,

NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

G. L., a white woman thirty-six years old, was brought to the Medico-Chirurgical Hospital in February of this year in semi-coma and unaccompanied by any one who could give an account of her illness.

She was very fat, weighing probably three hundred pounds. She was not, however, simply obese, because in addition to the uniform general increase in the sub-cutaneous fat there were great pendulous masses on the upper arms, the thighs and the abdomen which gave the sensation on palpation of a bundle of worms described by Dercum, and which were very sensitive to pressure, not only in the region of nerve trunks, but everywhere. The skin was not thickened nor adherent to the underlying tissue. In the skin over the masses there were many white, glistening superficial scars resembling those seen on the abdomen in women who have borne children. The hair, nails, and joints showed no trophic changes. There was no serous edema, no mucous edema, and no sign of acromegaly. The neck was so fat that it was impossible to palpate the thyroid gland.

She was, on admission, in extreme hebetude. She could with great difficulty be roused, but only for a moment. She answered questions, but only by single words, or if she attempted a sentence soon became confused and then silent and drowsy. She could give no account of her illness save to say that she had headache, felt sick, and could not walk, or see except dimly. Speech was slow, but not aphasic nor inarticulate. She

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\*Read by title at the twenty-sixth annual meeting of the American Neurological Association, May, 1900.

could not move either the arms or legs. She could flex and extend the fingers slightly, the toes not at all. She moved the head slowly and weakly, and swallowed what was put in her mouth, but made no attempt to chew. Sensory examination was difficult because of her mental dullness. Whether touch was felt could not be determined. Pain sense was preserved, and in the fatty masses there was hyperalgesia. The knee-jerks were very much diminished. The biceps tendon, plantar, jaw and abdominal jerks were absent. The condition of the muscle-jerks could not be determined on account of the fat overlying them. Vision was very bad, and there was marked optic neuritis. The thoracic and abdominal organs were normal. The urine at the first examination did not contain albumin, sugar, or tube casts, but later casts and albumin were present. Several days later I obtained the following fragmentary history from her brother: She had always been well till about two years ago, when she began to be heavy and dull, and changed from an active woman into a lethargic one. She began to grow fat several years before that. About six months before coming to the hospital she began to complain of weakness in walking. The difficulty slowly increased, and two weeks before admission she suddenly became palsied on the right side for a few hours. She then rapidly lost power in both legs and arms, and lost control of the bladder and rectum. She talked well until two weeks ago.

She remained in the hospital several weeks, continuing semi-conscious, paying no attention to her bladder or rectum, speaking only a word or two now and then, never moving the arms or legs, and swallowing liquid food without trouble; then the coma deepened, and she died with edema of the lungs and acute Bright's disease. At necropsy there was found a tumor of the pituitary body about as large as a walnut, involving the optic chiasm and penetrating upward into the ventricles. There was quite marked internal hydrocephalus. The thyroid gland was normal in size, and contained a concretion about as large as a small chestnut. The lungs were edematous. The ovaries were small and hard.

My friend, Dr. D. J. McCarthy, hardened, sectioned, and examined pieces from the various organs. The fat revealed, on gross examination, a peculiar, fibrous character, it being firmer than normal subcutaneous fat, and showing here and there in encapsulated areas softer and less yellow in color. These isolated areas of fat varied in size from a pea to a hazelnut, and were surrounded by a distinct fibrous capsule. There appeared to be no connection between this fat and

the firmer fat imbedded in the fibrous structure surrounding the fatty nodes. These nodules were not immediately beneath the skin, but deeper in the subcutaneous fat, and could not have been distinguished by palpation. On microscopic examination the firmer masses treated with connective tissue stains revealed a marked increase of this tissue, whereas the fatty nodule was normal in this regard, but an artery which ran through was distended and its coats were thickened. We were unable to find any nerves in the fatty nodules, but in the surrounding fat and in the underlying muscles the smaller nerve branches showed a high grade of in-



Fig. I.—Nerve twig in subcutaneous fat, showing high-grade interstitial neuritis. Oc. 3; ob. 7.

terstitial neuritis. In the muscles this was associated with a granular fatty degeneration of the muscle fibers, the granules staining black with osmic acid. The interstitial changes in the fat nerves were more marked. The nerve fibers, when present, were few in number and surrounded by a dense connective tissue. In the nerve filaments in the muscle the connective tissue was decidedly increased, but there were more nerve fibers present than in the fat nerves.

The ulnar, median, sciatic, peroneal and tibial nerves

were examined. The Marchi, Weigert and carmine stains revealed no degeneration. In the sciatic nerves fewer nerve fibers were present than usual, and there was an infiltration of fat, most marked in the central areas, which gave to the fresh nerve a semi-gelatinous consistence.

The Brain.—A glioma the size of a walnut occupied the seat of the pituitary body and embraced the optic chiasm and nerves. The tumor extended into the interior of the brain, grew through the aqueduct of Sylvius, completely filling it and infiltrating its walls to a slight degree, and filled

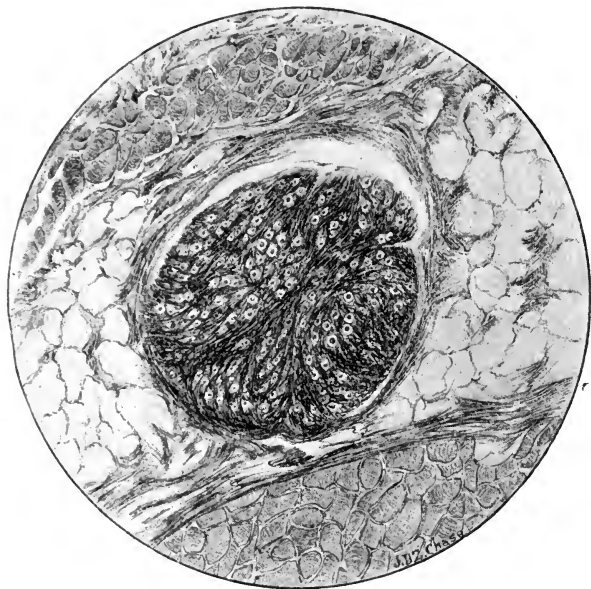


Fig. 11.—Nerve twig within the muscle, showing interstitial neuritis. Oc. 1; ob. 7.

up the upper area of the fourth ventricle. There was marked internal hydrocephalus.

The spinal cord showed a considerable grade of hydromyelia, extending down to the dorsal region. The external lateral group of cells of the anterior horn showed a light grade of chromatolysis.

In the right lobe of the thyroid gland there was a spherical concretion as large as a hickory nut. The gland was normal in size. On microscopic examination colloid degenera-

tion with atrophy and absence of the secreting cells in many acini was found. There were many areas of small round cell infiltration and indications of an active inflammatory process were present everywhere. The muscles showed marked degeneration with the osmic acid method. It was probably secondary to degeneration of the nerves, as a medium grade of interstitial neuritis of the intramuscular nerve bundles was present.

There was acute parenchymatous nephritis with slight low-grade interstitial change. There was marked fatty infiltration of the liver.

The ovaries were examined by Dr. H. L. Williams, who reported as follows: "Surface smooth. No inflammation apparent. Tunica albuginea appears very much thickened, sclerotic and hyaline. The deeper tissue of the stroma is dense and contains large numbers of primordial Graafian follicles, and in the connective tissue beneath the ovarian stroma are large numbers of blood vessels with well-developed muscular walls. Scattered here and there through the stroma are a number of corpora fibrosa and also a few small cystic Graafian follicles. Projecting from a surface in one portion is a small body the size of a split pea, the tissue of which is identical with that of the ovarian stroma with which it is directly connected. Its surface is smooth, but inflamed, and it contains, a short distance below the surface, a number of undeveloped Graafian follicles, and just at the surface a small cystic follicle. The specimen is a small, typically sclerotic ovary."

Any one who had seen the original patients of Dercum could have made the diagnosis of adiposis dolorosa in this case with certainty and without difficulty. The picture was classical. But just as certainly there was some organic brain disease in addition. Hebetude and, indeed, continuous stupor are not uncommon in the last stages of adiposis dolorosa, but transient hemiplegia, a rapidly oncoming palsy of all four extremities, and optic neuritis cannot be caused by it. The symptoms showed that there was local brain disease of such a nature as to cause variations in the intra-cranial pressure, but it was impossible at the time I saw the patient to determine the situation of the lesion. Post mortem it was found in the pituitary body.

The case is of interest, because it throws some light upon

the morbid anatomy of the disease, or at least shows what lesions are found. I know of no other necropsy in this disease, save the one which Dr. Dercum reports at this meeting. In general, his and my findings are the same—in both there is disease of the thyroid gland and of the finer nerve branches. But notwithstanding this agreement it is far too soon to have any fixed opinion as to the causation of adiposis dolorosa, or as to what organ is primarily at fault. The whole question is still in the region of hypothesis. Many more cases must be studied in life and death before safe conclusions can be drawn. Meanwhile in the present state of knowledge the thyroid gland is naturally first thought of as the organ disease of which would be apt to produce such symptoms. That disease of the thyroid gland profoundly affects nutrition is proven by myxedema and cretinism. The opinion that it may cause adiposis dolorosa rests rather on analogy than proof. Thyroidal concretions are not infrequently found accidentally post mortem and surely frequently do not cause symptoms of any kind. Much of the disease in the thyroid in my case was acute and hence could not have caused a condition which had begun years before. There were some pathological changes of long standing, however, and they may have had some relation with the adiposis dolorosa.

The most striking lesion was the tumor of the pituitary body. It completely destroyed all the glanular structure in that organ. This should have produced, according to a quite widely prevalent view, and according to my own published opinion, acromegaly. It did not. Whether the disease in it was a mere coincidence, or whether there was any causal relation between the tumor and the adiposis cannot be decided. It probably was coincidental, because in no other case has there been any evidence that the two conditions were associated—in no other case have there been present the symptoms of interference with the functions of the optic chiasm or the general symptoms of brain tumor. There may, however, in the cases studied only clinically have been atrophic disease of the pituitary body, and this would not cause symptoms which would attract attention to



it. One thinks of the pituitary body only when there is acromegaly or the characteristic hemianopsia. Post mortem it is neglected unless there is some very patent lesion.

The ovaries are interesting. They were sclerosed and not performing their great function. Ovaries and testicles have something to do with fat formation. Spayed or castrated animals are almost always fat. Eunuchs are most often obese. Women at the menopause often put on much flesh. It is a physiological incident. Artificially induced menopause is often followed by obesity. But all these conditions are mere fatness—a very different thing from adiposis dolorosa. At present we can only say that adiposis dolorosa is a clinical entity having definite signs and symptoms, but with an as yet unknown pathology.

107 LA MYOCLONIE EPILEPTIQUE (Epileptic Myoclonus). Rabot (Thèse de Paris, 1899).

The author states that Prichard was the first to describe this association. Delasiauve and Herpin were familiar with the phenomenon, as were other epileptologists. It has often been confounded with other affections, as by Hammond. Russell Reynolds stated that motor troubles of some sort occur in three-quarters of all epileptics. Recent authorities are cited by name, including some not usually enumerated (Weiss, Orazzio).

Nothing is more variable than the symptomatology of myoclonus in epilepsy. Féré has described bizarre movements in deglutition, and in the action of the diaphragm, producing abnormal respiration. Rabot also includes phenomena reported by the present reviewer (Dide) as belonging to myoclonus. Whatever occurs interparoxysmally of the nature of sudden repeated movements, having something of the quality of an electric shock, belongs here in Rabot's opinion. Rabot states positively that myoclonus, as an occasional symptom of epilepsy, is *not* the same affection as paramyoclonus multiplex, fibrillary chorea, myotonia, electric chorea, tic, hemiathetosis, etc. He would prefer to call it *petit mal moteur*, a manifest phenomenon of epilepsy proper.

CLARK.

# DISCUSSION ON THE NEURONE DOCTRINE IN ITS RELATIONSHIP TO DISEASE OF THE NERVOUS SYSTEM.

(Continued from page 514.)

## THE FUNCTIONAL SIGNIFICANCE OF THE SIZE AND SHAPE OF THE NEURONE.

HENRY H. DONALDSON.

### ABSTRACT.

These observations were made upon growing nerve cells in the white rat, as they appear between birth and maturity. In the growing spinal ganglion of the lumbar nerves, the increase in volume of the largest ganglion cell-bodies was shown to be very closely correlated with the increase in the area of a cross section of the nerve fiber growing out of these cell-bodies. The examination of the fibers was made on the peripheral side of the ganglion.

Further study of the cross section of the nerve fiber showed that the area of the axis cylinder was almost exactly equal to the area of the surrounding medullary sheath. This held true from the time that the medullary sheath was completely formed on the largest fibers of the sciatic nerve.

In the new-born rat, the sciatic fibers are totally unmyelinated, and there is a short preliminary period in which not only the axis cylinder increases, but the medullary sheath is added, and so rapid is this first formation of the sheath that in a few days it attains the relation just mentioned. The relations just mentioned are shown in the table which follows:

### WHITE RAT.

| Body Weight.<br>Grams. | Ratios.                 |             | Areas Axis<br>and Sheath. |
|------------------------|-------------------------|-------------|---------------------------|
|                        | Volumes Ganglion Cells. | Areas Axis. |                           |
| 4.7                    | 1.0                     | 1.0         | 1.0                       |
| 10.4                   | 1.6                     | 1.4         | 2.8                       |
| 25.7                   | 4.9                     | 4.6         | 9.3                       |
| 68.5                   | 11.2                    | 12.2        | 24.0                      |
| 159.0                  | 15.0                    | 14.4        | 29.7                      |

This equality in the areas of the sheath and axis persists through the entire growing period of the white rat. There are thus two phases in the growth of the medullary sheath, represented first by its rapid appearance, and second by its subsequent slow enlargement. It was shown from the data of

comparative anatomy that the *length* of the nerve fiber from a given cell was not correlated with the volume of the cell-body, and was apparently a matter of small significance; the increased length of the fiber not putting a direct nutritional tax on the cell-body itself.

In this connection, the observations of Dr. Elizabeth Dunn were also presented. These showed that the current dictum that the nerve fibers of larger caliber had the longer course did not hold true in the case of the nerve fibers supplying the thigh of the frog, where it could be demonstrated that the average diameter of the fibers innervating the thigh was greater than the average diameter of the fibers passing beyond the knee, to innervate the remainder of the leg. Moreover, the branches going to the thigh contained the very largest fibers that are found in the sciatic nerve.

This statement applies to the several physiological classes of fibers, taken all together, and does not distinguish between the afferent and efferent axones.

Finally, an interpretation of the caliber of the nerve fiber was attempted, and it was pointed out that fibers of large caliber tended to have an extensive terminal distribution. Where the nerve elements were few in number, as compared with the mass of skin or muscle to be supplied, this indicated a coarse innervation. If, however, the number as well as the size of nerve elements was large, a very fine degree of innervation might result, as in the case of the extrinsic muscles of the eye.

#### DISCUSSION.

Dr. J. J. Putnam said that there was one point that had not been brought out, which is perhaps a corollary from what had been said, namely, that we should modify, if not abandon, the notion that the cell-body has in any special sense the function of storage of impressions. If the cell-body is a network or meeting-place of fibrils, as has been urged, that view of its function is more probably correct which assumes that when any part of the brain is active a series of thrills passes through its component fibers from end to end, and the assumed "storage" should rather be rendered as changes in co-ordination. The sensori-motor mechanism acts as a whole, and there is no storage in the cells, which are rather "nodal points."

Dr. F. W. Langdon thought that to begin at this date to criticise the neurone doctrine is very much like criticising the utility of a building of which the plans are not yet drawn. We have all realized the truth of the saying that facts are stubborn things. He thought we ought also to remember that they are not only stubborn, but exceedingly *useless* things until connected by something in the nature of theory or hypothesis, and, we may safely say, with regard to the neurone doctrine, that anatomically it has helped us to convert, perhaps, what was but a jungle or wilderness of foliage into something resembling an orderly garden of shrubbery and classified plants.

Pathologically it has enabled us to collect together a large number of otherwise disconnected and useless facts, which we have, in some measure, converted into a pathological mosaic of considerable practical value.

Clinically, we were very much in the position with respect to the nervous system before the announcement of the neurone doctrine that the hematologist would have been before the discovery of the blood corpuscles. How would he have differentiated between the anemias? Therefore, to criticise at this early date the neurone doctrine as not leading to radical results in treatment is a little premature. It is our imperfect knowledge of the details of the neurone doctrine, not the principle itself, which is at fault. Even in this imperfect state the neurone doctrine has led not only to practical advances in anatomy, physiology and pathology, but also, by reason of furnishing a clearer basis for thought, to better results in diagnosis and prognosis; and it cannot fail to lead eventually to a more rational treatment of nervous diseases. If a man can think clearly he is more likely to intelligently treat a disease than if he argues from a disconnected mass of facts, as we formerly did. Dr. Langdon wished to go on record, therefore, as being firmly convinced of the great importance of the doctrine in a practical as well as in a theoretical sense.

Dr. Van Gieson said so much had been given in the symposium on the neurone theory that it was difficult to attempt any adequate discussion. We ought all of us to acknowledge our indebtedness to the first speaker for bringing together in his work on the nervous system in book form the scattered facts and theories of the neurone doctrine which have accumulated in the vast literature of the past decade. Works like those of the first speaker which bring together desultory facts and observations upon the neurone into a coherent whole, thereby giving an opportunity for viewing the interdependence and relationship of these facts are fully as important as the individual observations and discoveries, and indeed more valuable.

At the same time Dr. Van Gieson did not know that the neurone doctrine at this late day needed so strong a defense. The neurone doctrine now has become universal and valid knowledge; it is accepted as a well-grounded scientific principle, founded on an enormous supply of inductive data. The establishment of the neurone theory is overwhelming and to combat the older views, or a few recently found facts which seem to speak against the doctrine, seems like slaying the slain and vanquishing what is already defeated. The neurone doctrine is the extension of the universal cellular structure of tissues in general to the nervous system, and His in 1879 brought out this view in demonstrating that each axone was the outgrowth and dependency of the corresponding neurone cell-body. Quite true, it is not so long ago that students obtained the impression from such a text-book as Gray's Anatomy that nerve fibers were one thing and ganglion cells another. Dr. Van Gieson felt that all of us who have occasion to teach the anatomy or functions of the nervous system begin with the neurone doctrine as soon as possible and consider that the former views merely have a historical interest. What is there, then, that speaks against the neurone theory? He could not see how concrescence of neurones gives the slightest basis to doubt the validity of the neurone doctrine, or the primary character of the neurones as individual cells. We might as well question the individual character of the stellate branching cells in myxomatous tissue, because they anastomose, or any other cell which has intercellular bridges or anastomoses. This question of neurone concrescence in whatever character it appears is a secondary modification dependent upon the fixed nature of the function of the annectant neurones. Nor could Dr. Van Gieson see how the exposition of the neurone fibrils gives us the barest foundation to doubt the neurone theory and that the neurones are primarily individual cells.

In regard to the splendid array of facts brought out by the second speaker the suggestion may perhaps be timely that we shall not be able to understand all these morphological data of the neurone until we devote more attention in connection with structural data to the function and physiology of the neurone. If we make use of the parallelism of fluctuation of neurone energy with the succession of psycho-motor phenomena, our interpretation of these phenomena as well as of structural neurone changes is greatly illumined. The rise and fall of the threshold of neurone energy, it seemed to Dr. Van Gieson, is really one of the greatest ideas in interpreting a great host of phenomena in abnormal nervous life. These morphological data are liable to remain in a chaotic condition until we use

them in connection with some general guiding principle underlying the phenomena of neurone function.

The third paper at the beginning brings some disappointment to the hopes that ultimately the extension of the neurone doctrine into the domain of abnormal mental and nervous life would bring an increase in our power to cure or control these manipulations. It appears, if Dr. Van Gieson understood the speaker, that from a practical standpoint incurring disease we are not much better off than we were before. This in general may be quite true, but we need not be discouraged at the slow fulfilment of gain in practical treatment born of the neurone theory. We have hardly begun to apply the neurone theory. Dr. Van Gieson meant more especially the physiological neurone theory and not the morphological theory—to unravel the succession of morbid nervous and mental phenomena. But the extension of the doctrine into this field is sure to revolutionize our whole conception of clinical neurology and psychiatry. While it is quite true that some stages of the dissipation of neurone energy are beyond restitution, one thing is sure, and that is, that we cannot handle a mechanism until we understand it; we cannot lay hold of phenomena until we know their succession, and can forecast them. To work out the succession of phenomena the establishment of laws and theories are requisite. This being the case, we are somewhat surprised to hear the speaker sound a note of warning that theory after theory is being piled up on the neurone conception. We can have no ultimate progress of the neurone doctrine in the treatment of nervous and mental diseases without these theories. Far from being a disadvantage, calling for rebuke, these theories are, on the contrary, of the greatest help. Facts without theories is chaos. We need fear no apprehension about the growth of theories of the neurone principle. The inadequate ones in the course of time will be weeded out and do no harm, while the broader theories which stand the test of verification will undergo the selection of the fittest and eventually stay and become valid knowledge. For his part, he had had occasion to witness the direct outcome of the neurone doctrine in actual practical cures of the most signal importance, in psychopathic functional disease. We are working in this direction all the time and our great guide and mentor are some psychopathological principles, that we have worked out from the neurone conception. As for the retraction theory he was more firmly convinced of its validity than ever. Ameboid motion of the neurones had been mentioned here this morning. We ought to get rid of this term and the idea it conveys. This notion seems

to have grown out of Wiedersheim's observations which Dr. Van Gieson felt sure time will prove to be erroneous. There is no such coarse expression of neurone motility as ameboid motion would convey. Only the terminals of the neurones may be supposed to contract and expand, and this indeed to a slight and perhaps almost infinitesimal degree. Two years ago, and again since that time, Dr. Van Gieson had expressed the view that the nervous system is neither made up of conrescent nor free neurones exclusively, but of both. And, to him, it seemed strange to see a sharp line of division established in the discussion of neurone retraction, one school having it that neurones are conrescent, the other that they are retractile. If one would stop to reflect on the subject a little, it would be obvious from the nature of nervous and mental phenomena, both normal and abnormal, that both types of neurones go to make up the nervous system. The field of the microscope is not the place to prove neurone retraction. If he should actually be able to see evidences of neurone retraction under the microscope, it would not add one iota to his belief in its existence, any more than his belief in the molecular theory would depend upon his catching hold of a molecule and holding it in his hand. Neurone attraction we assume, and if it is verified by psychomotor phenomena its proof is just as strong as the verification of the molecular hypothesis by the phenomena of the physical world. The people who demand actual perceptual demonstration of neurone retraction before believing in it seem to forget that the very nature of a hypothesis is such as to transcend the limitations of our perception. Hypotheses are conceptual creations, but always similar to and congruous with our perceptual data.

In fact, while they were ardent champions of the retraction theory as applied to the higher functions of the nervous system, Mr. Weil and Mr. Frank working in the Pathological Institute had shown that the data of the objective demonstration of neurone retraction under the microscope is utterly unreliable. It is the deductive application of neurone attraction that establishes our basis of belief in this theory, and with Dr. Sidis a preliminary argument was presented under the title of "Neurone energy and its psychomotor manifestations" sustaining this view.

The fourth paper is a veritable treasure. Here is the ontogenetic source of data which fit in perfectly with the laws of neurone cytolysis, disaggregation of neurone hierarchies—except that in ontogenetic, and probably also in phylogenetic development, the phenomena are presented in reverse order of those of neurone cytolysis.

Dr. F. X. Dercum said that notwithstanding all that has been said, he did not know of any theory that offered as good a working hypothesis as regards the explanation of mental processes, both normal and pathological, as does the retraction theory. He did not for a moment maintain, however, that actual physical retraction of cell processes is necessary; indeed, he could conceive of the changes consisting of mere physiological interruptions in the relations of the neurones to each other without gross physiological change. However, he thought it is best for us to wait until our knowledge on the subject has been more complete, until we know more about the details of the relations of the neurones to each other in all portions of the nervous system, before attempting a further explanation. He had learned the danger of speculating on unknown grounds. Still a working hypothesis has its value. For instance, no theory of memory that has ever been offered can even approximate that offered by the so-called retraction theory. It is better to wait, however, and the old German adage may still be true—"He who laughs last, laughs best."

Dr. E. B. Angell said that Dr. Barker had touched on one point that we as clinical neurologists have been waiting for, from the laboratory workers of the day, the question of the nutrition of the neurone. We have been too much taken up with the study of the morphological changes of the neurone from a histological standpoint, and while he appreciated the work of Dr. Spiller he thought a great deal can be learned from the study of the influence of circulation upon the changes in the peripheral neurone. In a case he had seen a short time ago an arterio-fibrosis was associated with multiple neuritis affecting the feet. In one foot gangrene had resulted and amputation of the foot had been resorted to. The surgeons had been compelled in turn to amputate the toe, the ankle, and finally the foreleg. The neuritis was not recognized and on account of the arterio-sclerosis the nutrition of the peripheral neurone was faulty, in consequence of which the gangrenous condition reappeared at the higher level.

Dr. Angell suggested large doses of nitro-glycerine, knowing that we could in that way get some dilatation of the fibroid arteries. Nitro-glycerine had been tried, but with insufficient strength. Under the influences of large doses the local nutrition was improved and the patient recovered. The neuritis was in time relieved and the man is well to-day. Such is the clinical evidence of the importance of studying the local nutrition of the terminal neurone, and he believed if some proper methods could be devised by laboratory workers for studying the local circulation much light could be thrown on the changes in the



neurones, not only in the terminals of the neurone, but also in the cell-bodies.

Dr. J. J. Putnam said that this differentiation between the cell-process and cell-body which had been referred to by Dr. Spiller is important from a pathological point of view, and also from a physiological point of view. We all remember the interesting experiment of Prof. Bowditch, showing the nerve fiber is incapable of fatigue, whereas the nerve cell is readily fatigued; so that the cell as a whole is a differentiated body, and that lays additional weight to what Dr. Sachs had said, that in dealing with pathological and clinical cases, we should have a record of facts as they are, and not force them to suit anatomical conceptions. We should not forget the debt that we owe to the old notion of "trophic domain," of which Dr. Barker had spoken. The neurone theory has as yet given us little else beyond the reinforcing of this notion that is of practical value in clinical pathology.

Dr. Joseph Collins said that he was somewhat bewildered, as well as very materially instructed, after listening to this discussion. In discussing the neurone doctrine we are not dealing with theories, but with facts, and, in the solution of many of the questions that come up in pathology and in physiology, we are endeavoring to fit pre-conceived theories with the facts of the neurone doctrine. It is always a difficult matter to discuss papers constituting a symposium, because they supply a complete and final discussion from different points of view. We are under obligation to the first speaker for having presented to us in such a clear, succinct, and critical way, this complete digest of the present status of the neurone doctrine. Its value was quickly manifest in the reading of the following papers, pointing out distinctly the necessity of using the term nerve cell as synonymous with neurone, and not speaking of the nerve cell when the cell-body is meant, and other clear distinctions.

Dr. Collins said he had long endeavored to follow Dr. Van Gieson and Dr. Dercum, as they had striven to unravel the contraction theory, and it seemed to him that until something more convincing was forthcoming we should devote no further time to it. Dr. Collins congratulated Dr. Spiller on the amount of pathological and anatomical material which he had been able to bring to corroborate his statements, and to make lucid demonstrations of the tenets which he had held. Dr. Sachs, he said, had taken a somewhat more pessimistic view of the bearing of the neurone theory on the explanation of nervous disease than Dr. Collins was inclined to take, yet he felt in sympathy with much that had been said. Up to the

time that the neurone doctrine was brought forward, Dr. Collins had had no consistent and satisfactory idea, as he had now in a measure, of the different forms of progressive muscular atrophy and dystrophy, including amyotrophic lateral sclerosis. But it seemed to him now that he could use his pictorial memory in explaining the various forms of dystrophy and atrophy, whether of the Aran-Duchenne type, or the neural or muscular variety, by the aid of the neurone doctrine.

Dr. L. F. Barker said that in the very interesting series which Dr. Spiller brought forward of partial degenerations of the neurone, he (Dr. Barker) was reminded of the occurrence of partial cell degeneration elsewhere in the body. We can have partial cell necrosis in the liver or kidney cell without causing complete death of the cell.

As to the relation of the pyramidal tract to the ventral horn of the spinal cord the observations of the actual changes in the cell-bodies of the lower motor neurones are extremely interesting. They have a bearing on the question of the exact relation of the upper to the lower motor neurone. Many will recall that von Monakow has maintained that the axones of the pyramidal tract do not come into direct connection with the cell-bodies of the ventral horn cells, but that there is a group of small neurones (Schaltzellen) interposed between the upper motor segment and the lower. It will be interesting to follow these pathological changes and to see whether ultimately we shall have to consider the cortico-muscular conduction path as consisting of two sets of superimposed neurones or of three.

The volume of the axis cylinder of the nerve fiber as compared with that of the nerve cell body had interested Dr. Barker for some time. His attention was first forcibly directed toward it by reading Dr. Donaldson's book on the growth of the brain. It is, if he remembered correctly, in that that he makes the statement that the volume of the axis cylinder may be one hundred and eighty-seven times greater than the volume of the cell-body.

Dr. Barker wished to say that his conception of the neurone was not based on purely morphological grounds, but just as much on physiological and pathological results as upon form relations.

With regard to the relation of the neurone doctrine to the study of disease clinically, he had been glad to hear that clinicians have been as much helped as they have. It was surprising to him that the doctrine should have entered so quickly into clinical thought.

Dr. W. G. Spiller said he had understood Dr. Donaldson to say that in the same animal the axone supplying a distal

part of the limb was not larger than that supplying a proximal part, that is, the diameter of a long axone might be smaller than the diameter of a shorter axone. He was not quite sure that he understood Dr. Donaldson's method of determining this fact. Dr. Donaldson had taken the axone of a nerve in the thigh, for example, and found it to be of a certain diameter. It seemed to Dr. Spiller possible that an axone in the thigh may give off collaterals, and that this axone may become smaller as portions lower in the limb of the animal are examined. He had not understood whether Dr. Donaldson took into consideration the distribution of the collaterals from the portion of the axone in the upper part of the limb.

The explanation of atrophy in cerebral disease, Dr. Spiller thought, was very difficult. It has been believed by some to be due to arthritis, by others to alteration in the cells of the anterior horns of the cord, and by others to vascular changes produced by cortical lesions.

Dr. Spiller said that he was becoming more and more skeptical as regards the importance of chromatolysis without other lesions. Chromatolysis is of value in determining the location of the cell-bodies of diseased axones, but as indicative of altered function it has a limited value. The displacement or destruction of the nucleus, the tumefaction of the cell-body, the destruction of the dendritic processes, the alteration of the other contents of the cell-bodies, seemed to him more important than the breaking up of the chromophilic elements, and it has been shown by experimental work that chromatolysis may occur without any disturbance of function having been noted during life.

Dr. Sachs remarked that there was evidently an impression abroad that he was not especially fond of theories. He did not think less of theory, but more of facts. There is a vast difference between a theory and a fancy. The theory must always be developed *pari passu* with the facts. What would we have thought of the Darwinian theory if Darwin had not furnished that stupendous array of facts in support of his teachings no one has been able to deny. Do not let the theory jump ahead of facts, and don't waste time to find the evidence in support of the theory when you can look through the microscope. The evidence must be practical and more spontaneous. Dr. Sachs thought that the neurone theory would do a great deal in the further development of our understanding of the nervous system. If he had seemed pessimistic, it was because he was anxious not to indulge in fanciful pathological discussions.

Dr. H. H. Donaldson said that Schwalbe started with the theory that the largest nerve fibers have the longest course.

He next developed the idea of the conical diminution of the fibers in their course. That was necessary to support the doctrine, because the large nerve fibers were not to be found at the periphery. If they run a long course, then they must disappear on the way. Conical diminution was brought in to account for this disappearance. That is Schwalbe's statement of the situation:

Dr. Donaldson had evidence that in the course of the sciatic nerve, where the branches are given off for the innervation of the thigh, that approximately 6 to 8 per cent. of the fibers given to the branches are the result of division of fibers, one portion of which runs on below the knee and the other stops in the thigh. That the diminution in the average diameter below the knee is due to a conical diminution is met by the observation that in the branches to the thigh the maximal fibers are practically all found. These appear above the point at which the branches leave for the thigh and they appear in the branches which are distributed to the thigh, and thus their course is accounted for through its entire extent.

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108 "UEBER DIE VORÜBERGEHENDEN ZUSTÄNDE ABNORMEN BEWUSSTSEINS IN FOLGE ALKAHOLVERGIFTUNG, UND ÜBER DEREN FORENSISCHE BEDEUTUNG" (Transitory Abnormal Conditions of Consciousness Due to Alcohol Poisoning and Their Forensic Indications). Moeli (Allg. Zeitschrift für Psychiatrie, 1900, LVII, 2 & 3, S. 169).

The author makes an important clinical contribution to the study of temporary mental disturbances due to alcohol poisoning. The histories of fourteen cases are studied in detail and their close connection with alcoholic indulgence is shown. It is pretty clearly established that they are not to be regarded as examples of true epilepsy. A number of them developed on an hereditary basis; in others intolerance to alcohol appeared after an accident of some sort. Most of them had been guilty of criminal acts, ranging from simple exhibitions to violent assault and even to murder. The medico-legal points involved are considered at some length by the author.

ALLEN.

REVISED INTERPRETATION OF THE CENTRAL FISSURES OF THE EDUCATED SUICIDE'S BRAIN  
EXHIBITED TO THE ASSOCIATION IN 1894.\*

BY BURT G. WILDER, M.D.

Abstract.

After an interval of six years this case is again submitted to the Association upon two grounds:

In the first place, it has a potential medico-legal aspect inasmuch as upon the determination of the identity of the fissures depends the accuracy of the description of the wounds; secondly, I have taken for granted that there always will be rejoicing among neurologists over the frank admission of an error.

As indicated in the title of the paper, I have seen reason to modify the interpretation of the peculiar fissural condition of this brain which was offered to this Association in 1894.<sup>1</sup> The brain at that time had recently come into my hands under very peculiar circumstances, and I was perhaps unduly impressed by them. The individual, Dr. W. I. Brenizer, was a dentist who had willed me his brain in writing, and who shortly thereafter killed himself by two successive pistol shots. The first ball merely abraded the mesal aspect of the left hemisphere and did not prevent him from putting a second through his right temple. This traversed vital parts and emerged near the middle of the convexity of the left hemisphere, in the course of a long fissure having the general direction and aspect of the central or fissure of Rolando.

But in the effort to describe the wound of exit more particularly I encountered difficulties due to unusual conditions. Caudad of the wounded fissure is one not so nearly reaching the dorsal margin. Cephalad are two, one dorsal, the other ventral, slightly overlapping, but separated by a distinct

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\*Read at the twenty-sixth annual meeting of the American Neurological Association, May 1, 2, and 3, 1900.

<sup>1</sup>Exhibition of a suicide's brain with two pistol-ball wounds; remarks on its fissural anomalies. *Amer. Neurol. Assoc. Trans.*, 1894, 102-104; *JOUR. NERV. AND MENT. DIS.*, Dec., 1894; *Congress Amer. Phys. and Surg.*, III, 24; *New York State Med. Soc. Trans.*, 1894, 190-194.

isthmus. Similar conditions exist on the right side, excepting that the two left disconnected fissures are evidently represented by a single continuous one.

About that time I had become very much interested in the central fissure, and had recently encountered the description of Giacomini of what he considered a case of duplication of it. Calori is said to have reported a similar case. At any rate, I fell under the fascination of the idea that I had been the fortunate obtainer of a third case of duplication of the central fissure, and at the time of the presentation of the paper in 1894, and up to comparatively recently, I still maintained that the conditions were more easily explained upon the hypothesis of duplication than any other way.

At first, of course, it might appear that the two separate fissures in front of the wounded one could not represent either *the* central fissure or one of two such, but rather a pair of pre-centrals. But the brain of Chauncey Wright, which has been shown to the Association, and of which I have photographs here, has a completely interrupted central fissure on both sides. Several similar cases are recorded. There is no impossibility in interpreting the two as a central fissure on account of their separation—firstly, because they correspond to the single fissure on the right side, and, secondly, because there are cases in which the central fissures are more or less completely interrupted. At any rate, I did come to the conclusion provisionally that we had here, as in Giacomini's case, a pair of central fissures on each side, with this peculiarity, that on the left the cephalic or anterior one was interrupted. As to the junction of it with the Sylvian, that is so common that nothing need be said.

Since that time I have gone over my whole collection of brains, and have examined several in other collections. I have more than 100 well preserved human brains, and I have compared them all with reference to this point:—Is there any absolute diagnostic feature of the central fissure on the one hand, and the postcentral on the other, by which we can discriminate and identify them? So far as my material goes there is no feature of the human brain which is more constant and more

worthy of consideration than this, that the central fissure either indents the dorsal margin of the cerebrum or approaches it in such a way as to constitute itself a kind of arrow in connection with the bow formed by the paracentral fissure on the mesal aspect of the hemiserebrum; in other words, the characteristic feature, and one that is absolutely constant, so far as I am aware, of the dorsal end of the central fissure of man is to aim itself across the mesal aspect of the hemiserebrum, so as to cut into the paracentral gyre.

With nearly the same constancy the end of the paracentral fissure, indenting the margin, aims at the middle of the notch formed by the diverging branches of the dorsal end of the postcentral.

These characteristic topographic relations are not always obvious when the hemiserebrum is viewed from the dorsum, the meson, or the lateral aspect; but they are easily recognized when the dorso-mesal margin is nearest the eye, and there are visible, foreshortened, both the mesal and part of the lateral aspects, as in the photographs and diagram exhibited.

Applying these two tests to these two fissures, I come very reluctantly to the conclusion that we have here a central which is complete and approximately normal on the right side, but which varies from the normal in being completely interrupted on the left; and that the wounded fissure is not a second central, but rather an unusually long and complete postcentral.

This brain presents certain peculiarities, some of which might serve as arguments against the present interpretation; but, on the whole, this now seems to me less unsatisfactory than that which was offered six years ago. I shall be thankful for suggestions upon any of the points involved.

#### DISCUSSION.

Dr. F. W. Langdon asked Dr. Wilder how soon death occurred. The second ball, which, he believed, Dr. Wilder stated was sufficient to cause death—how soon did it cause death, and what was the immediate cause of death—was it hemorrhage or was it the shock or the hydro-dynamic effect of Horsley? What stopped, the heart or respiration?

Dr. B. C. Wilder replied that the whole thing was done in strict privacy in the man's office. He locked himself in his office. The deed was done at midnight and no one had any expectation of the act. Nobody knows what occurred. The action had always impressed Dr. Wilder as rather extraordinary for a person who had willed his brain. His persistence was remarkable. Is there any other case on record where a man, unless he was a maniac, tried to kill himself with one pistol ball and, failing, used a second?

Dr. H. H. Donaldson said that it was possible that the relation of the two gyri would go far towards determining this point. Of course, Dr. Wilder knows—he referred to the fact—that the posterior gyrus rather overlaps the anterior, the central fissure passing in, in an oblique manner.

The second point which Dr. Donaldson suggested is the fact that in some cases it ought certainly to be possible to take out a slice of the gyrus and subject it to a simple microscopic examination. The character of the cortex both for the pre-central gyrus and the post-central gyrus is so characteristic that a determination could be easily made as to the arrangement of the cells in the cortex, and this would give a basis for a morphological interpretation. It is possible that Professor Wilder feels an interest in not using other than certain methods by which to determine his conclusions, and Dr. Donaldson would not intrude on that artistic feeling. But the application of histology would be of great assistance in a question of this sort.

Dr. W. G. Spiller asked Dr. Donaldson whether he had found that the cells of Betz are not present in the upper and anterior part of the parietal lobe, adjoining the post-central convolution.

The parietal lobe in this anterior and superior part is a portion of the motor cortex. Dr. Spiller would like to know whether Dr. Donaldson had not found that that part of the parietal lobe is very similar in histological structure to the postcentral convolution in its upper part.

Dr. H. H. Donaldson replied that he could not answer the question in a way that would be of any use.

Dr. A. Meyer suggested the same point with regard to the determination of these fissures that Dr. Donaldson had mentioned. It seemed to Dr. Meyer that we are too much inclined to draw conclusions from a section out of the paracentral lobule, as we know that at times we fail to find Betz's cells in this region, while in other parts of the paracentral lobule, even down to the corpus callosum, we at times do find Betz's cells. Dr. Meyer would not insist so much on the presence of



Betz's cells as on the configuration of the whole cortex bordering the fissure at the lower end of its upper third. He thought it would be relatively easy to see there that the anterior central has a typically "motor" arrangement, whereas in the posterior central we find the transition into the structure which is commonly called the structure of the parietal cortex.

Dr. Chas. K. Mills said that the report of Dr. Wilder's second case emphasizes the importance of not coming too quickly to a decision that we have a duplication of fissures. He had examined many human brains during the last twenty years, and, while perhaps he could not say frequently, he could say that in quite a number of instances he had seen an appearance of duplication of the central fissure quite as marked as in this brain presented by Dr. Wilder. We should never conclude that we have the duplication of a main fissure like the central until we have excluded all other explanations of the apparent duplication.

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109 EIN FALL VON SYSTEMATISCHER ERKRANKUNG DER SEITENSTRÄNGE BEI CARCINOME, KLINISCH UNTER DEM BILD DER SPASTISCHEN SPINAL-PARALYSE VERLAUFEND (A Case of Systemic Disease of the Lateral Columns in Carcinoma, Appearing Clinically as Spastic Spinal Paralysis). E. A. Meyer, (*Deutsche Zeitschrift für Nervenheilkunde*, Vol. 16, Nos. 5 and 6, p. 345).

Meyer describes a case of carcinoma of the uterus in which he observed a spastic paretic gait and exaggerated patellar reflex and Achilles clonus on each side. No motor paresis when the patient was examined in bed; no ataxia, no disturbance of defecation or micturition, or of sensation were discovered. In the lumbar region only the crossed pyramidal tract was degenerated, but in the thoracic and cervical regions the adjacent part of the direct cerebellar tract was involved. The degeneration did not extend above the cervical swelling, and was most pronounced in the lumbar region, and decreased in intensity towards the cervical cord. It was not the so-called descending degeneration, for the brain was intact, and no transverse spinal lesion was found. The rest of the cord was normal, and it is especially stated that the spinal gray substance was not diseased. The carcinoma of the uterus was believed to have been the cause of the degeneration of the pyramidal tracts, probably through toxic products.

SPILLER.

## RIGIDITY OF THE SPINE.

By J. H. McBRIDE, M.D., Los Angeles, Cal.

The following notes of cases may be interesting in connection with the subject of the paper by Drs. Sachs and Fraenkel in the JOURNAL for January.

These notes were made thirteen years ago and are now published as then written. In the light of later experience I could wish they were fuller and more accurate. The illustrations are from photographs made at the time.

Case I.—Man, single, age 48, soldier during the war of rebellion, was not wounded; no history of syphilis or hereditary disease. In 1865 had pain for some weeks in left big toe; a few months later left hip began to get stiff and this continued to trouble him slightly for years; later the stiffness increased. In 1876 his spine began to get stiff. His left arm was paralyzed for a few days at one time and recovered and then got gradually stiff. Later his right arm and hip were involved. Since then his spine, including neck, has gradually become stiff, and the entire spinal column is now but slightly movable.

On examination patient stands with body leaning slightly forward. The spine is held in fixed position, the head being thrown forward and carried in this position apparently with great care. He can turn his head to either side only to the slightest degree, the chin deviating perhaps an inch, not more. If he wishes to see anything when sitting, he turns his entire body. Knees are slightly bent in apparently from contraction of adductor muscles; hip joints are almost immovable. If he stands on one foot and attempts to swing the leg, the movement of the leg is very limited and the pelvis moves with the leg. All muscles of the spine and leg above knee are rigid. Those of shoulder and arm above elbow somewhat rigid, but less so than leg; shoulder movements greatly limited by rigidity of muscles; cannot put hand to head. If he wishes to pick anything from the floor, drops on knees. Can reach his feet to tie shoes only by getting on his knees and reaching his shoes behind. All movements of arm below elbow and of leg below knee are normal or nearly so.

Except the pain mentioned in left foot, occurring early in disease, has never had any pain, spasms or tremors; skin of back markedly hyperesthetic; knee-jerks exaggerated; slight ankle-clonus on both sides. On tapping any spinal muscle or

the deltoid or one of the serrati a contraction of the muscle occurs. There is no atrophy of muscles.

The dorsal spine from first to eleventh is slightly curved to right, the deviation being one and one-half inches in deepest part of curve. Patient says the spine was stiff for a long time before curvature developed. There seems to be some tenderness over the part of the spine that is curved, though this is



Case I. Position in standing and walking.

doubtful. The curvature seems to be due to the unequal pull of the contracted muscles.

Patient can be placed in a chair with some difficulty, for owing to the stiffness of hip joints he practically lies on his back in the chair. In walking leans forward, steps cautiously and carries head and body as a solid, inflexible mass.

In 1876 sight of left eye gradually failed, and in a few years he was able only to see light. In 1885 sight in right eye began to fail and he can now only read large print. Hearing is getting poor in both ears. Has occasional attacks of left facial neuralgia.

Action of bladder and rectum normal. The left optic nerve is atrophied. So far as I could determine with the ophthalmoscope the right optic nerve was normal. He was never examined by an oculist.

Though the following case of spinal rigidity does not apparently belong to the same type as the first one, yet it seems worthy of publication. My diagnosis at the time was hypertrophic cervical pachymeningitis. As it was traumatic it is probable that there was injury to the spinal column itself.

Case II. J. C., an Italian laborer, age fifty-four. No history of syphilis or of any previous illness, or of any hereditary tendency to disease.

In June, 1887, one year ago, fell into a large hole, striking his forehead on a stone, his feet resting on the opposite side of hole, his neck being thus badly strained; was unconscious for some time (does not know how long); confined to bed for three or four weeks, his neck being stiff and painful. A little later arms got weak; muscles wasted, and he had pain in back of neck and arms, and a sensation of ligature around arms at insertion of deltoid.

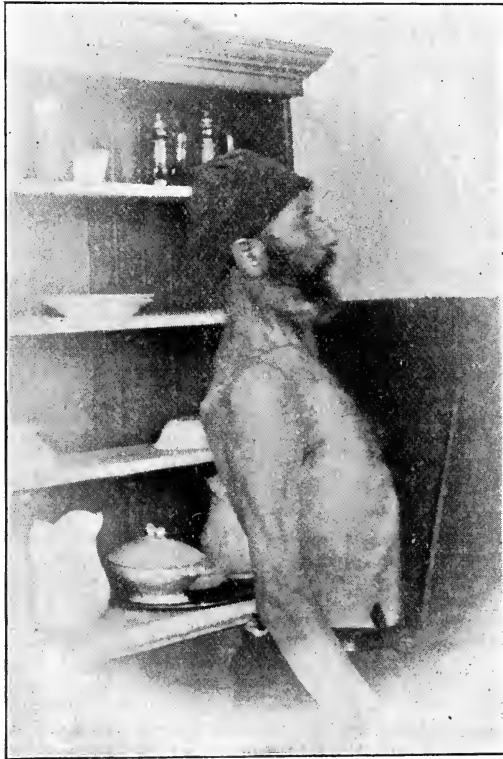
Examination in June, 1888: Patient holds his neck in fixed position. Cervical and upper dorsal spine rigid. Lower dorsal and lumbar spine apparently normal. The only movement possible in the neck is a slight nod of the head, and even that is painful. All muscles of back, neck and shoulders much atrophied; anatomical outlines of scapulæ plainly visible. Muscles of both arms atrophied, the wasting being less below the elbow. Movements of left hand greatly limited; those of right less so. This is seemingly due to paralysis, atrophy and rigidity combined. Fingers of left hand held semi-flexed; left arm hangs by his side practically useless. Can carry right hand to opposite shoulder with effort, and with greater effort carries same hand to his mouth.

Pressure over fifth cervical vertebra causes pain, and pain is felt in same locality if forcible effort is made to turn his head. At present has no pain in arms or legs. Muscles of legs atrophied and apparently somewhat parietic with slight rigidity. There is also some rigidity in all arm muscles.

He walks with shuffling gait slowly and cautiously, and

strikes his toes on floor as the foot is brought forward. Dynamometer, right 10, left 6. No discoverable tactile impairment, though he says fingers feel dead. No thermo-anesthesia, no paresthesia, no loss of muscle sense. Bowels constipated; passes urine normally.

Examined in December, 1889. Disease has made steady progress. Has feeling of constriction around body, about on line with eighth rib. His gait is peculiar. Walks in quick jog-



Case II. Habitual position one year after accident.

ging steps, moving his foot only a few inches at a time, his head bent forward, neck rigid, trunk bent far back, arms extended and hands crossed on body in front. Face has an anxious frightened look. Has some pain in region of 5-7 cervical vertebræ. Legs cramp and jerk at night and also during the day if he lies down. No fibrillation of muscles.

Superficial and deep reflexes all exaggerated. Ankle-clonus marked. The slightest tap on any muscle will cause the muscle to contract. All muscles react to induced current. The biceps is more atrophied than the triceps. Extensors of forearm more atrophied than flexors. Breathing entirely pectoral. When bowels are loose, as they frequently are, they move involuntarily. Urine has been passed involuntarily for some months. Eyesight good for age. Fundus normal.

The painful period that Charcot and Joffroy have described as part of the clinical history of hypertrophic cervical pachymeningitis was not pronounced in this case. Rendu has, however, shown by an autopsy in one case that the dura may be involved only in its anterior portion. In such case posterior root symptoms would be absent, and they would, if present, correspond in intensity to the degree of involvement. Some allowance, perhaps, should be made for the social level of the individual, for a stupid and tough-fibred laborer would not feel pain so acutely, nor recall so well the pain he had felt, as a more delicately organized and observant person.

I did not see the patient during the last six months of his life, and do not know if the contractures of limbs described by Charcot developed during the last stages of the disease.

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110 UEBER ACUTE PARANOIA (Acute Paranoia). M. Köppen (Neurol. Centralbl., 18, 1899, p. 434).

There exist wide differences of opinion among authors regarding this affection. Although some consider possible the existence of a curable acute paranoia combined with hallucinations, yet others declare the disease to be incurable and state at the same time that the condition described as a curable paranoia should be ascribed to other forms of disease. The progress of the disease alone can hardly be taken as a criterion for the assumption of the presence of psychoses. The name "paranoia" is applied to a diseased condition in which delusions are prominent, while there is no neglect of the personal condition manifest. Köppen says that these attributes are also to be found in acute cases. Very often, indeed, he states, based upon a ground-work of some pathological condition, such as imbecility, hysteria, epilepsy or even dementia paralytica and senilis, we find engrafted the picture of paranoic symptoms. But in cases where no such ground-work exists, it is perfectly proper and fit to make the diagnosis of *paranoia acuta*. The disease may begin with a general condition of excitement or mind-wandering. The delusions and illusions are like those of chronic paranoia. In imbeciles and alcoholics these latter show a characteristic coloring.

A CASE OF PRIMARY PROGRESSIVE MUSCULAR DYSTROPHY OF THE FACIO-SCAPULO-HUMERAL TYPE OF LANDOUZY AND DEJERINE.

BY ALLAN BLAIR BONAR, A.B., M.D.,  
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Cases of this form of dystrophy are sufficiently rare in occurrence to warrant the presentation of a case without many comments.

The case here reported is that of a young Jewish girl, 17 years of age. She has been, and is still, a worker in artificial flowers. Her parents are living, and as far as they know, there has been no paralysis of any sort in the family. Heredity seems good. About eight months ago the patient noticed that she could not put her right hand to her head. This was the first and only symptom complained of by the patient, and the one for which she came to the Vanderbilt Clinic. She says, however, that she has always been very thin, which condition is accounted for by the probable existence for some years of the atrophy of the muscles of the shoulder girdle; she has always considered this thinness to be general. Furthermore, she says that she has never been able to close her eyes completely, nor to smile as other girls did.

On examination it is found that all the muscles of the shoulder-girdle are atrophied except the infraspinati. The pectoral muscles are almost entirely gone, only a few fibers of the clavicular portion of the pectoralis major remaining on each side. There is a little more of this clavicular portion remaining on the left side than on the right. The trapezii are both affected, but the right trapezius is much more atrophied. The latissimi dorsi are weak, as shown by the peculiar gait, or rather, by the throwing back of the upper part of the body in order to balance herself when walking or sitting. The patient says that her friends have spoken of, and she herself has noticed lately, this weakness of the back and the tendency to lordosis. The muscles of the upper arms are affected, the deltoid, the triceps, and the biceps, each being atrophied. As in the case of the other muscles of the shoulder-girdle the atrophy

of the muscles of the upper arm is much greater on the right side than on the left. The scapulæ, especially the right, are drawn high up on the shoulders and rotated, so that they have become "winged scapulæ." The patient can raise the left arm to the horizontal position, but not the right, unless the scapula is held fixed to the dorsum. Because of the atrophy of the pectorals and the position of the scapulæ the patient presents a very hollow-chested appearance. The atrophy of the muscles of the shoulder-girdle is much more marked on the right than on the left side.

In the face the orbicular and buccinator muscles are chiefly involved. The weakness of the orbicularis palpebrarum is shown by the inability to close the eyes completely. The involvement of the orbicularis oris allows the lips to protrude and produces the "tapir mouth." The patient cannot whistle or blow out the cheeks. The weakness of the muscles around the mouth is further shown by asking the patient to separate the lips and show her teeth. This she accomplishes only by pushing the lips apart with her fingers. This patient presents the typical "myopathic face" due to the weakness of the orbicular muscles. When speaking the cheeks of the patient puff in and out, showing a weakness of the buccinators, but she says she has no difficulty in eating or in managing food in her mouth. When she smiles it is seen that the risorius and zygomaticus major on both sides are much affected, inasmuch as the angles of the mouth are not drawn outward and upward as in a normal smile, in fact the mouth hardly moves at all.

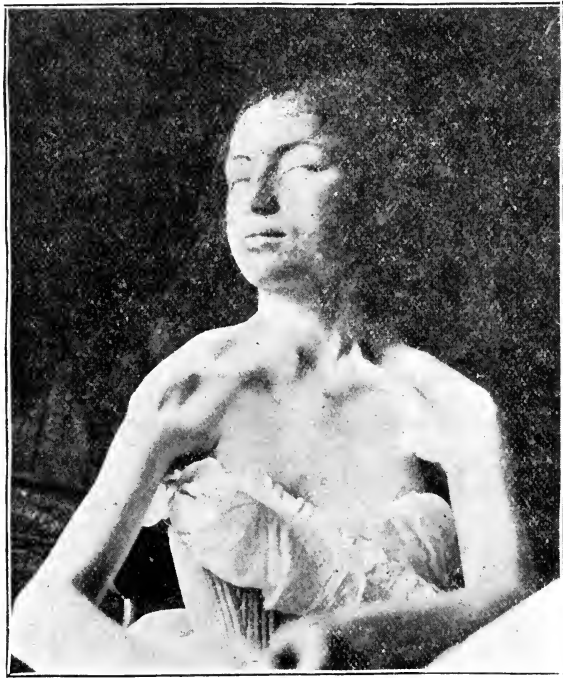
The muscles of the hands and forearms are unaffected and are not atrophied, and the patient works regularly making artificial flowers. The muscles of mastication, deglutition, and also those of the eye, are not affected; neither is the diaphragm, nor the other muscles of respiration as far as can be ascertained.

The lower extremities are not involved at all, though the patient says that her friends have noticed a limp on the right side. This is probably due to the greater weakness of the muscles of the back on that side, but has not been observed by the writer. The lower extremities are well nourished. There is no hypertrophy of any of the muscles of the body.



The reflexes are diminished, the knee-jerks being obtainable only by reinforcement, and then barely perceptible. The right knee-jerk seems slightly less active than the left. The wrist and elbow-jerks are present, but scarcely perceptible.

The accompanying photographs, for which I am indebted to Dr. Walter Timme, illustrate very well the inability to close the eyes completely, the "tapir mouth," the "myopathic face,"



and the atrophy of the shoulder-girdle muscles. In the photograph showing the face, the blemishes are due to an accident to the plate.

Although the patient and her mother have been questioned very carefully, absolutely no history of any paralysis or atrophy in the family, or of any other hereditary factor, has been obtained.

This type of dystrophy usually begins in childhood at about

the third or fourth year, and the weakness and atrophy appear first in the face. The statement of this patient that she has never been able to close her eyes completely, and that her smile was always peculiar and different from that of other girls, shows undoubtedly that her trouble began in the face and at a very early age. As is usually the case, the progress of the disease has been very slow, and the atrophy of the shoulder-girdle



muscles has not been considered as anything except ordinary thinness until the weakness in the shoulders produced a disability. Although this patient's thighs and legs are well developed, the atrophy in the face, chest and upper arms probably gave her the idea that she was thin all over. This patient presents a remarkably typical case of progressive dystrophy of the Landouzy-Dejerine form.

## A CASE OF HYSTERICAL APHONIA IN A GRAND MAL EPILEPTIC.

BY L. PIERCE CLARK, M.D.

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The occurrence of hysterical aphonia is not rare, but the condition occurring in an epileptic is not only uncommon and interesting, but is also quite unique. The case is as follows:

Mr. J., aged 31; German; with a bad family history. Epilepsy of grand mal type began at 25 years of age, and has had attacks of petit mal and grand mal about every three weeks since. Patient is of fair mental and physical condition. He has been under constant observation for the past four years. A history of a condition of temporary aphasia after convulsions was given by patient's sister. Shortly after coming under my observation, patient had a severe seizure followed by entire loss of speech. He was able to whisper a few words but did not recover from the aphonia for six days. It was thought that the condition might be a post-convulsive exhaustive aphasia, and careful attention given to hysterical stigmata did not disclose the well-known symptoms of that disease. The diagnosis of post-convulsive exhaustion of the speech center was allowed to stand until several attacks of aphonia had followed petit mal. At last the condition occurred independently of convulsions, and then strong suggestion was sufficient to re-establish the full power of speech in a few minutes, and upon suggestion he has become aphonic again. Power of speech has been restored after all sorts of epileptic phenomena, so the diagnosis of post-convulsive epileptic aphasia, can be no longer held. The patient loses speech only at certain hysterical nodes which the convulsions seem, in the majority of instances, to determine and accentuate.

Only in the absence of the hysterical stigmata in aphonia following grand mal epileptic seizures, can we be sure of the exhaustive nature of the lost or disordered speech. Strong suggestion or even hypnosis must also be tried. Undoubtedly many cases heretofore supposed to be based upon exhaustion, or inhibition according to Gowers and Lowenfeld are

really conditions of hysterical aphonia. Isolated or combined hysterical phenomena are to-day found to be of much more common occurrence in connection with typical epilepsy than was formerly thought to be the case.

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III EPILEPSY ASSOCIATED WITH PARAMYOCLONUS. Verga and Gonzales (Annali di nevrologia, 1900, XVII, 6).

These authors quote an authority, Bresler, who found that all his myoclonics were epileptics, and who advanced the theory that paramyoclonus was a spinal epilepsy. The authors' own cases are as follows:

Case I.—Male, single, aged 32 when he entered the asylum in 1884. Had been in similar institutions four times since 1875. His work, when he was employed, was heavy, and he depended on alcoholics for energy. One brother and two sisters were epileptic. His epileptic attacks began at the age of seven. At the age of 20 the myoclonus began. His character is very irritable, culminating at times in mania. His epilepsy is not of a severe type, about one attack per month. The myoclonus affects the limbs and face, and consists of a mild tremor and twitching of muscles, to which are occasionally added more violent jerking motions of head and limbs. Patient is now 49 years of age. He exhibits numerous marks of degeneration, asymmetry of face, nose deviated to left, absence of lobules of ears, vicious implantation of teeth, etc. Special senses and pupils normal, and in general he passes a good physical examination. The principal anomalies are the myoclonus, which affects the integrity of voluntary muscles, the gait, etc., the epileptic attacks, and the mental state, consisting of weakness of mental faculties with irascibility.

Case II.—Male, entered asylum in 1882, at age of 32, where he remained up to 1895, when he died. Epileptic since the age of 10. Four out of ten brothers and sisters are epileptic, two of the four having also paramyoclonus. His epilepsy was of a grave type, attacks being both frequent and severe. The paramyoclonus began at the age of 16. The patient originally passed a good physical examination, and his senses, reflexes, etc., remained normal. His myoclonus was incessant and interfered with all voluntary acts, such as eating and drinking. Perhaps for this reason he gradually passed into a state of marasmus.

Case III.—Male, admitted to asylum in 1884, at the age of 19. Epilepsy and myoclonus came on simultaneously at the age of 12. Several brothers and sisters likewise affected. The patient's epileptic attacks were not frequent or severe in type. The myoclonus, however, was incessant and severe. The mental state was one of weakness and irascibility. General physical condition was unimpaired. Patient died in 1889. A fibrinous exudation was found on the arachnoid, at the base of the brain; miliary granulations on pia,—tuberculous, meningitis.

The cases occurred in degenerates, all with strong family history of predisposition. CLARK.

PHILADELPHIA NEUROLOGICAL SOCIETY.

April 23, 1900.

The President, Dr. Wm. G. Spiller, in the chair.

PERIPHERAL FACIAL PALSY WITH CROSSED HEMI-PARESIS.

Dr. F. Savary Pearce presented a colored man, teamster, aged 58, with crossed paralysis, in whom the hemiparesis seemed in some way connected clinically, at least, with the complete peripheral paralysis of the seventh nerve. The man came to the clinic of Dr. J. Madison Taylor at the Howard Hospital on the seventeenth of June, 1899, presenting complete left facial palsy. There was no trouble with the musculature of the limbs at that time. He stated that three days previously he noticed that he could not close his left eye, the complete facial paralysis rapidly supervening. It is likely the real paresis began four weeks earlier when he sat in a draught for some time. This exposure was followed by pain in the left parietal and mastoid regions, with tenderness on pressure over the same areas, existing in some measure up to the present time. He was treated by blistering and potassium iodide in five grain doses three times a day, and was referred to the ear clinic where supuration of the left middle ear was diagnosed and treated by Dr. Vansant. There was no disturbance of sensation over the paretic area, nor was there any disturbance of the sense of taste, but the tip of the tongue was pushed toward the right when protruded. The man was treated carefully with descending galvanism during the summer which seemed to restore in part the paretic facial muscles. There has never been more than the slightest alteration of the formula as evidence of degeneration of the left facial nerve. In October, 1899, Dr. Posey examined the eyes and found the discs normal and no changes in the fundi. There is, at the present time some tenderness over the left mastoid region, but the ear has ceased discharging.

The man was admitted to the ward in October and remained under galvanic treatment with continued use of iodide of potassium and 1-20 grain doses of strychnine for seven weeks. There has been practically no permanent improvement in the face.

On December first, in the morning, while walking upon the street he became dizzy, nauseated, and finally vomited much bile. He then became dazed, and had to be carried home. He was found to be feeble in the right leg and arm and was unable to walk for some time. He came to the clin-

ic again on February 24, 1900, at which time both knee-jerks were found to be increased, especially the one on the right side; the right arm and leg were feeble, and the tongue was slightly protruded toward the right side.

Dr. Pearce thought that the cause of the left facial paralysis was septic otitis media with extension and perhaps permanent impairment of the seventh nerve. The sudden onset of hemiparesis of the right side of the body, three months later than the beginning of the left Bell's palsy, suggested to him that an abscess had developed in the left motor region of the cerebrum secondary to the ear disease; or that a primary hemorrhage involving the left motor area of the brain had occurred. Although there were no symptoms of abscess of the brain, Dr. Pearce thought that a pus collection extending by contiguity from the septic left ear might be the remote cause of both paralyses.

Dr. Charles W. Burr thought that the most natural explanation was that the man had Bell's palsy, and, later, an apoplectic attack. Hemiplegia from abscess is not very common, because an abscess is not usually in the motor region.

Dr. F. X. Dercum remarked that Bell's palsy as an independent affection in hemiplegia is not very uncommon, and abscess almost never gives rise to hemiparesis.

Dr. William G. Spiller agreed with Dr. Burr and Dr. Dercum that the crossed paralysis was probably the result of two different lesions. Abscess from ear disease is more commonly in the temporal lobe, and if hemiparesis had occurred by the extension of such an abscess to the internal capsule, we might expect to find symptoms of sensory aphasia in this case, the lesion causing hemiplegia being on the left side of the brain. He referred to a case seen by Dr. C. K. Mills and himself, in which there was paralysis on the same side as the facial palsy, developing at a different time. A hemorrhage into the optic thalamus had occurred, and the paralysis of the facial nerve was an independent condition. In this case, the center of the facial nerve afforded an interesting study, as it was possible to distinguish by the altered cells the side of the pons belonging to the affected facial nerve.

#### A CASE OF ACROMEGALY.

Dr. W. G. Shalldcross, by invitation, presented a male of eighteen years with acromegaly. The disease had only been recognized for about two and a half years, though the history showed that it may have had an earlier origin. The symptoms began to develop after an acute illness.

He was born in normal labor, and was the first child of his parents. His father was below the average in intelligence, and was deaf. The patient has one living brother, normal in every respect. In August, 1892, he was described as being of the usual weight and size for his age. His head was of normal shape, and his features were long. In walking he bent for-

ward, and in speaking stammered a little. He commenced to walk when sixteen months old.

In 1892 he entered the Pennsylvania Training School, where he made fair progress, and soon learned to read and write, and was regularly promoted from time to time. His mental development continued until April, 1896, one year previous to the onset of the symptoms of acromegaly. By this time he had reached the highest grade in the Pennsylvania Training School, which is attained by only a small minority of those under instruction. From now on until the present time his mental faculties have progressively lessened.

In 1897 he began to grow rapidly, and the peculiarity of growth which is so pronounced now was then noticed. From the average build of a boy of fifteen years of age up to the present time, in three years, he has grown to his present extraordinary development. The hands are symmetrically enlarged and out of proportion to his body. The bones and soft parts have shared equally in this hypertrophy. There is apparently no thickening of the bones at the articulations, and the hands present the so-called spade-like appearance of the English, or the battledore head of the French. Fleshy pods on the palms, so often described in this disease, are well shown here. The fingers are long and sausage-shaped. The feet correspond in the main to the description given of the hands. They are, respectively, right, twelve and one-quarter inches; left, twelve and one-half inches long, and he wears what is known as a number 15 shoe. The face is strikingly characteristic. Its oval contour, the long and broad nose with dilated nostrils, the thick lips, massive malar bones and lower jaw give to it a decided prognathism. The lower jaw has grown forward about one-quarter of an inch in the past year, so that the normal articulation is lost.

In speaking the voice is noticeably altered. It has the peculiar deep resonance that is so frequently heard in patients suffering from acromegaly. The tongue is long, broad, and deeply fissured, and seems unwieldy; in appearance it resembles closely this organ as seen in the cretin or the Mongolian type of imbecility. The soft palate and uvula are correspondingly enlarged. The larynx is rather large, and the neck thick. The right thyroid lobe seems to be distinctly enlarged and quite firm.

The spine shows a dorsal kyphosis which dates back several years, at least, before the disease developed. The thoracic cage bulges forward, and the antero-posterior diameter is increased as compared with the lateral. The abdomen is pendulous and

the breasts markedly enlarged. The sternum and clavicles are hypertrophied. This increase in the size of the latter is particularly noticeable on their inner thirds. The ribs are broad and flat, and at their junction with the sternum are noticeably beaded. The bony framework seems everywhere enlarged. As the patient stands in his stocking feet he is six feet three and one-half inches in height, and when stripped weighs 200 pounds. In a little more than a year he has grown one and one-quarter inches in height. The weight, however, has remained the same.

Hearing shows no change as tested by watch; right ear, six inches; left ear, sixteen inches. Smell is normal; taste is also normal.

His gait is peculiar, and is largely due to the tilting of the pelvis, caused by deformity of the right knee and the effort at maintaining equilibrium, made difficult on account of the kyphosis.

His station and sway are normal. Cutaneous reflexes and knee-jerks are diminished. His strength has progressively failed. Grip, as tested by dynamometer: right hand, 40; left, 35. Hyperhidrosis, so common in these cases, is more marked on right side. The skin is appreciably thickened, and over his shoulders, sides of chest, and lateral surfaces of abdomen it is thrown in rugæ.

Dr. Francis X. Dercum said that this was undoubtedly a case of acromegaly in which there was associated gigantism. It suggests the thought that when acromegaly begins in early life, before the epiphyses and shafts of the bones have become united, there may be increase in the length as well as the breadth of the bones. The case he considered a very interesting one, although not as typical as some, probably because it was in course of development.

Dr. Charles W. Burr reported a case of treadler's palsy.

Dr. John K. Mitchell referred to a similar case he had seen in which there was cramp as well as palsy in the posterior muscles of the lower leg. It was a comparatively acute form, the man having been working only a few months. He was completely cured by rest and galvanism.

Dr. A. A. Eshner said that a similar disorder had been observed in motormen from excessive ringing of the gong with the foot.

#### A CASE OF AMYOTROPHIC LATERAL SCLEROSIS.

Dr. Charles S. Potts presented a case of this disease in which the especial points of interest were the possible influence of lead in the etiology, the unilateral distribution, and the rapid development of the symptoms.

The patient, a man 42 years of age, had worked in lead works for eighteen months. Nine months ago he had an attack consisting of pains in the stomach, and constipation, which his



physician called lead colic. About six months ago he noticed weakness of the left leg and his toes caught in any object projecting above the level of the floor. Very shortly after this the left arm became weak, and at about the same time indistinctness of speech and difficulty in swallowing were noticed. He had not observed any weakness of the right side of the body, but examination showed it to exist.

Dr. James Hendrie Lloyd said that in its rather recent onset, and in many of its clinical aspects, this case resembled one which he had recently had in the Philadelphia Hospital. His case began acutely last fall with symptoms of irritation of the spinal meninges. When admitted, one month ago, the patient was found to have amyotrophic lateral sclerosis, with a spastic gait and ataxia, marked fibrillation, and slight muscular atrophy, most marked in the essential muscles of the hand.

The lesion of the cord seemed so acute that it suggested the propriety of lumbar puncture for the purpose of making a bacteriological study. Two punctures were made with practically negative results.

In Dr. Potts' case lead may have had something to do with the causation. The symptoms of lead poisoning are not confined to the classical wrist-drop. Well-marked cases of muscular atrophy have been reported in persons who have long been exposed to the action of lead. He had himself reported one such case in the article on occupation neuroses in the "Twentieth Century Practice."

Dr. Francis X. Dercum alluded to the existence of bulbar symptoms in this case, and referred to a case of amyotrophic lateral sclerosis reported by Dr. Spiller and himself, in which bulbar symptoms were present. He commented upon the frequency of bulbar symptoms in amyotrophic lateral sclerosis.

Dr. William G. Spiller called attention to the interesting fact that in Dr. Potts' case the disease began as an unilateral affection. Usually when a case of amyotrophic lateral sclerosis comes under observation the paralysis is symmetrical on the two sides of the body, but cases with unilateral symptoms are on record. In Dr. Potts' patient the disease had a rapid development. Dr. Spiller had recently exhibited a case of amyotrophic lateral sclerosis in which the symptoms had not progressed during two years.

Dr. Charles W. Burr presented a woman with general anesthesia, who was blind and deaf, and whose movements were very inco-ordinate.

Dr. Charles K. Mills asked where Dr. Burr thought the meningitis and meningoencephalitis were located. He referred to a case in which the autopsy showed meningitis and meningoencephalitis of both postero-parietal regions. This man had anesthesia of both halves of the body, but not general anesthesia. He had other symptoms, such as athetoid movements and some hyperesthesia.

Dr. F. X. Dercum said that an important point was that there was no pain and no motor involvement. It was strange that only a general surface anesthesia with involvement of the optic and auditory nerves should be produced by a meningitis.

Dr. William G. Spiller suggested that the lesion might be in the optic thalami and the tracts from the corpora quadrigemina passing

close to the optic thalami. The headache, vomiting, optic atrophy, and other symptoms might be explained by involvement of the optic thalami. Loss of hearing might be produced by involvement of the tracts from the posterior colliculi of the corpora quadrigemina to the temporal lobes. Such a diagnosis could only be held as a possible one.

Dr. Charles K. Mills was inclined to doubt the diagnosis of meningitis in this case. He thought that the lesion was a more localized one involving the sensory tracts or the thalami.

Dr. Charles W. Burr did not wish to be understood as asserting positively that this woman had meningitis, although he thought this diagnosis explained the symptoms. The exact nature of the lesion, he thought, could not be determined unless an autopsy were made.

#### A CASE OF RHIZOMELIC SPONDYLOSIS.

Dr. F. X. Dercum briefly placed on record the following case which apparently belongs to the group of cases described by Marie and Astie, and others, in which progressive rigidity of the spine is the most prominent symptom. Simple spinal rigidity may be the result of various morbid processes. Thus in the affection described by von Bechterew as *lepto-meningitis* with involvement of the spinal nerve roots is the essential lesion. In another group of cases the vertebral column is especially involved, and in such cases the affection is probably allied to chronic rheumatoid arthritis. The following case appears to belong to this second group.

C. S., female, aged 17, a native of Pennsylvania. Father and mother in good health. One sister dead of pneumonia. Three sisters and two brothers alive and well. Paternal grandmother died of pulmonary tuberculosis. The patient was weak and undersized in childhood. Had scarlatina and parotitis at an early age. Puberty occurred in the fourteenth year. Menstruation was at first irregular, becoming regular in the fifteenth year, too frequent in the sixteenth, regular again in the seventeenth year, and during the last year the periods were sometimes delayed, but never painful.

Two years ago the patient while walking across the floor developed a sudden pain in the left knee. She "walked lame" for several weeks, and was placed in bed with "rheumatism" for a month or more. After four or five months she began walking with crutches. The leg finally became apparently well and she walked normally from September, 1898, until April, 1899. At that time pain developed in both groins and a physician was again called in. She was not obliged to go to bed, however, until June, 1899; she has remained in bed ever since.

On admission to the hospital the entire body was found much wasted. The patient laid on either side with both thighs flexed on the abdomen. There was marked fixation in adduc-

tion at both hip joints, and the right knee could not be completely extended. There was excessive hyperesthesia of the entire abdomen with rigidity of the abdominal muscles. The area of hyperesthesia began above at the eleventh rib and extended around the body obliquely downward to an inch above the umbilicus, and was apparently symmetrical and extended to both groins and as far down the back as the end of the sacrum. The buttock, thighs and legs were not hyperesthetic. There was very marked hyperesthesia along the spine from the dorsal region down, and this was especially marked in the lower dorsal, lumbar and sacral regions. Deep pressure over the spine produced great pain. The knee-jerks were both preserved but greatly diminished. The pupillary reflexes were normal. The shoulders and cervical spine were not involved. The sphincters were normal. Examination of the urine was negative.

#### A CASE OF INTENSE INTERNAL HYDROCEPHALUS.

Dr. W. G. Spiller exhibited the brain from a woman who had been an inmate of the Pennsylvania Training School for Feeble Minded Children. The patient was sixty-two years of age, and had been under the care of Dr. Llewellyn, from whom the clinical notes were obtained. She had been a sickly babe, and was said to have been hydrocephalic from birth. Sight, hearing and speech were good. She had had epileptic attacks for years, and in these frothed at the mouth and had involuntary defecation but during the last six years of her life none of these attacks were observed. She occasionally had vertigo or vomiting. No motor paralysis was ever detected, excepting that she walked as though she were lame. She was a fairly intelligent person. The ventricles of the brain were greatly enlarged and the cortex and adjoining white substance were much atrophied, and measured in thickness scarcely more than a quarter of an inch. Sufficient cerebrospinal fluid was collected to nearly fill a quart basin, and yet some escaped. The absence of feeble mentality and paralysis in a case of such intense atrophy of the brain was noteworthy.

Dr. Charles K. Mills said that this specimen reminded him of the first case of hydrocephalus which he had examined post mortem, a case at the Elwyn Training School. The man lived to the age of about 50 years, and had pretty fair preservation of brain power. The brain presented much the condition seen in this specimen. It is interesting to consider how the brain adapts itself to the rest of the body and the rest of the body to the brain in these cases. He believed that the slow increase in the fluid and the slowly increasing compression of the tracts and the cortex of the brain, accounts for the preservation of the motor and other functions, as well as of some mentality, the brain accommodating itself to the slowly acquired new conditions.

## Periscope.

### CLINICAL NEUROLOGY.

- 112 **UEBER SENILE EPILEPSIE (Senile Epilepsy).** Redlich (Wien. med. Wochenschr., Mar. 24, 1900).

A hereditary taint often plays an important rôle in senile epilepsy. About one-quarter of all cases show distinct heredity (Mendel, Gowers). In genuine senile epilepsy there is at times a history of convulsions in infancy. Chronic alcoholism plays a most important etiological rôle as well. Injury to the head appears to be able to precipitate essential epilepsy, in some cases by mere shock. Acute and chronic infectious diseases, such as malaria and syphilis, are also etiological factors. Other elements entering into senile epilepsy are a high degree of atheroma of the blood vessels of the brain, arterio-sclerosis, and valvular heart disease, all of which may act as contributory causes in essential epilepsy. Cardiac and arterio-sclerotic epilepsy form a class by themselves, in which benefit is derived from the use of digitalis, caffeine, etc., and the bromides appear to be of little or no value.

In two cases of senile epilepsy, in which an autopsy was held, extensive general atrophy of the brain, chronic hydrocephalus, and pronounced arterio-sclerosis of the larger cerebral vessels were all absent; in their stead occurred only histological changes, "military sclerosis." In a third case, however, this condition was not present. Numerous others have found this gliosis in young subjects as well, but authorities like Binswanger have decided that its presence stands in no definite causal relationship to epilepsy. In the author's case there were several instances of serial attacks. Post-convulsive paresis, aphasia, etc., which are so common in the youthful, was noted in the aged. Therapeutically, there are no differences in senile and youthful epilepsy.

**DISCUSSION:** Lauterbach stated that the influence of organic heart disease in provoking genuine epilepsy was known in Delasiauve's time, and Schlesinger agreed with Redlich in his view that heart disease is an important factor in epilepsy. **CLARK.**

- 113 **APHASIE AMNÉSIQUE (Amnesic Aphasia).** Trenel (Nouvelle Icon. de la Salpêtrière, Nov., Dec., 1899).

In this article Trenel attempts to study more specifically the question of amnesic aphasia so thoroughly worked up by Pitrés. The latter believed that this form of aphasia was produced by the breaking up of a part of the commissural path, which unites the different centers of verbal images with that portion of the cortex in which the higher functions are localized. The study of Trenel was made upon two insane patients, who were affected with amnesic aphasia. The mental condition of both patients naturally made the psychological examination somewhat incomplete.

The first case is thus summarized: Woman aged forty-six years, amnesic aphasia with paraphasia. The amnesia is limited almost altogether to substantives. Word deafness present, but incomplete, no psychical deafness. Incomplete agraphia, hemiplegia with hemi-

anesthesia. Epileptic attacks. Circular form of insanity. Death due to cerebral hemorrhage.

*Autopsy.* The right hemisphere is the seat of a large hemorrhage, affecting all the neighboring parts and extending beyond the median line. There is no cortical lesion. Left hemisphere appears intact. A small hemorrhage is found in the first temporal convolution. A large hemorrhagic focus, in part cystic, in the middle part of the island of Reil, extending to the *pli-courbe* and limited to the white matter except at the point noted before. The two hemorrhages are almost symmetrical. A series of sections made by the method of Pitres shows the localization of the lesion to be sub-cortical. (Photographs of these sections are given in the article).

The aphasia is thus described: The patient knows what she wishes to say spontaneously and that which she should say in answer to the questions. The transference is formed irregularly, at times by abnormal paths, at other times by the habitual paths, parts of which have been destroyed. The aphasia of conduction best describes the condition. No definite localization could be determined in this case, but the lesion being confined to the white matter makes the theory of the sub-cortical origin of amnesic aphasia very probable.

The second case is briefly that of a woman fifty-nine years old. Incomplete hemiplegia on left side with hemicontracture. Generalized pareto-spasmodic condition. Dysarthria. Paraphasia. Amnesic aphasia. The patient cannot name most of the objects shown to her. She describes them, defines or indicates their nature. Her sentences are incomplete and badly constructed. No word deafness. Dementia, and great irritability. This condition approaches that of the first case and its localization is as doubtful. SCHWAB.

114 UEBER SPINALEPILEPSIE (Paramyoclonus and Epilepsy). Bresler (Neurol. Centralblat, 1896, XV, 1015).

History of brother and sister: I.—Female, age 26, consultation 1893. For past four years has been helpless, unable to earn living. Her disease began at the age of 6 years, at about the same time as her brother's, he being much older. There were first twitchings in face and limbs, next epileptiform attacks. Development good, no deformities. Mental faculties good. Face exhibits many scars from falls. During examination muscular twitchings increased in violence. Twitchings especially marked during voluntary muscular effort. Epileptic attacks very numerous, often several in a day. The back muscles twitch so violently that patient is nearly flung from her chair. The movements are like electric shocks. At present patient must be bathed, fed, and dressed. Patient much depressed over her condition. Careful testing of reflexes, special senses, pupils, conformation of bones, etc., shows no anomaly. Headache frequently occurs, with tender infra- and supra-orbital points. The muscles, so frequently in use, are not increased in volume, but seem soft and weak. There are "good" and "bad" periods of perhaps fourteen days' duration. Mental faculties now exhibit weakened memory and judgment, with irritability and quarrelsomeness. The author saw two epileptic attacks, one of which was mild in degree, and was immediately followed by movements like habitual twitchings. Convulsions, etc., were alike on each side. This patient died of hypostatic pneumonia. Autopsy:—Skull bones not thickened; dura of convexity adherent to bone; pia very delicate, transparent everywhere; vessels only moderately filled; at the apex of the frontal brain, on both sides, were two symmetrical

defects the size of a pea; ventricle not dilated; cerebral substance firm, uniform, but slightly moist; fatty heart, kidneys and liver.

II.—Brother of preceding, age 33. Normal development. Disease began when 12 years old, first with twitchings, later followed by convulsions and loss of consciousness. The twitchings gradually became so violent that patient was obliged to leave school. He is a large and powerful man of good mental power. The twitchings prevent speech to some extent. There is difficulty in stair-climbing. If anyone goes to his help the convulsions become worse. Many scars on face from falls during epileptic paroxysms. Sometimes he has several of these a day. In the course of time the patient's mind became quite weak, while in body he was helpless, and had to be fed and dressed. Finally, he developed some kind of fever and died with symptoms of paralysis of the brain. Autopsy:—No focal disease of brain; pia of central convolutions cloudy; convolutions, especially of occipital lobes, flattened; high degree of fatty heart.

Both patients gained considerably in weight under Flechsig's treatment, while the convulsions were brought to a standstill. Bresler concludes by calling this complication of epilepsy spinal epilepsy, or epilepsy of spinal origin. He quotes experimental evidence in connection with excitation of the cord (Brown-Sequard, Nothnagel, Unverricht).

CLARK.

- 115 PARECCHI CASI DI MIOCLONIE, LA MAGGIOR PARTI FAMILIARI (Cases of Myoclonus, Mostly Family Cases). D'Alloco (Riforma medica, 1897, I, p. 223).

The author has studied in all 24 cases of myoclonus, and of these 19 occurred in family groups. These cases are briefly described in detail. In a summary the author recapitulates each case and attempts to look upon a number of them as combinations of epilepsy with myoclonus.

First Family Group. Patient No. 1, aged 9 years, male. During the previous year received a blow on the head. One month later a rotary vertigo while at mass, lasting a few moments. A few days later a well-marked attack of petit mal with sensory aura, a fall and loss of consciousness. No mention of convulsions. A similar attack three days later. When examined it was observed that he was affected by clonic fibrillary twitchings, sudden in character, which at times provoked muscular movements of the limbs. These twitchings were especially marked on the abdomen, nates, sides, back, and chest; less marked on the extremities. They were increased with the emotions. Muscular strength well preserved. Reflexes diminished. Numerous stigmata of degeneration were present. The three sisters of the patient, all myoclonic, are not mentioned as having epilepsy. But the contrary is not stated either.

Second Family Group. First patient, a girl aged 10 years. First, she had persistent headache, then one month later, psychical disturbances, ecstasy, hallucinations, etc. There were also attacks of unconsciousness and falling, but no distinct convulsions. Several of these seizures occurred in succession. On examination the usual symptoms of myoclonus were seen to be present. Possible stigmata of degeneration, but intelligence superior. No evidence of psychical disturbances. The patient's brother, aged 14, is also a myoclonic—no mention of epilepsy—and is free from stigmata.

Third Family Group. First patient, a boy aged 5 years, presents numerous features of interest, his case being described at considerable length. Neuropathic inheritance. When he was eight months old

the mother observed that when the vertex was touched, the child being then sitting up, it would lose control of its head and fall over. If the face was touched, or even the hair, ever so slightly, the child would fall back, but without loss of consciousness. When seen by D'Alloco, the examinations covering a period of twenty days, the results of previous falls upon the head were evident. There was no doubt that an epileptogenous zone, involving the head and face, was present in the child. Upon this zone a tap with the finger, a slight pressure, or merely stroking the hair, caused a general instantaneous muscular shock, with relaxation of the limbs producing a fall. The author regards this case as a combination of reflex epilepsy with myoclonus. The boy's sister, aged 15, was slightly myoclonic. She was free from evidences of degeneration.

Under the head of general remarks, the author cites the opinion of Seppelli, Unverricht, and Bresler, as authorities on the association of myoclonus and epilepsy.

CLARK.

### ANATOMY.

116 THE TOTAL NUMBER OF FUNCTIONAL CELLS IN THE CEREBRAL CORTEX OF MAN, AND THE PERCENTAGE OF THE TOTAL VOLUME OF THE CORTEX, COMPOSED OF NERVE CELL BODIES, CALCULATED FROM KARL HAMMARBERG'S DATA; TOGETHER WITH A COMPARISON OF THE NUMBER OF GIANT CELLS WITH THE NUMBER OF PYRAMIDAL FIBERS. Helen Bradford Thompson (Journal of Comparative Neurology, Vol. IX, No. 2, 1899, pp. 113-140).

The estimation of the number of the nerve cells in human cortex cerebri is a problem as scientifically important as it is difficult. In this report appears what is undoubtedly the closest approximation to the fact yet published, and the chief product or result does not a little to further the doctrine of parallelism between consciousness and bodily structure in that *it increases eightfold the largest estimation previously made* regarding the number of these cells. This probable increase careful psychological analysis of the mental side long ago made more than likely, some sort of psychophysical parallelism being always (at least since Spinoza) more than probable.

Miss Thompson's method was simply to perfect the process employed by Hammarberg, Meynert, Wagner, and Donaldson, correcting by recent data their errors of judgment and supplying what they had obviously omitted. Prof. L. F. Barker also must be given the credit for supposing the largest previous estimation too small.

Hammarberg had divided the cortex for convenience of the enumeration into sixteen regions, and made six layers of cells, namely, nerve fibers with a few small scattered cells, small pyramidal cells, large pyramidal cells, small irregularly shaped cells, ganglion cells, and spindle-shaped cells. He employed as the unit-cube of measurement a bit of the cortex 0.1 mm. square, the number of cells in each of these in any particular region being taken as constant.

"If Hammarberg's records were complete in every detail, they should, then, contain the following data upon each of the sixteen regions into which the cortex is divided; first, the thickness of each of the six layers; second, the two diameters in micra for the average cells of each layer; and third, the number of cells in each layer contained in a cube measuring 0.1 mm. on a side. But not all of these data are given for every region. Sometimes the thickness of a layer is omitted; sometimes the size is merely indicated by saying that they are larger or smaller

than those of a corresponding layer in some adjacent region; and in some cases the frequency is not given. All these omissions have been supplied as accurately as possible."

The average number of cells in a column one centimeter square through the cortex was found for various places in each region, and this number varied between 29,880 in the sunken part of the temporal lobe and 76,615 in the exposed part of the occipital lobe. Donaldson's estimation of 2,352 sq. cm. as the total area of the cortex was employed, apportioned as follows: Frontal lobe, 41 per cent.; parietal, 21 per cent.; temporal, 27 per cent.; occipital, 10 per cent., and the island 1 per cent.

Without going into the elaborate and mathematically correct details or reporting any of the numerous tables of statistical results, we may here state Miss Thompson's estimate of the functional cortical nerve-cells in man as about 9,200 *millions* (about 0.23 per cent. of the number of cells by Francke's estimation in the whole human body).

The second part of the present research considers, as its product, that only 1.37 per cent. of the total volume of the cortex is made up of functional nerve-cell bodies.

The third and last portion of the paper makes an estimation of the cortical giant cells, and presents the number as about 159,690, while the pyramidal fibers for the limbs and trunks combined are about 158,222. As the difference (only about 1,500) is not large enough a number for the pyramidal fibers supplying the head and neck, it is concluded by the researches that these latter fibers take rise in other cells located in the cortex, probably in the large pyramidal cells in the fifth layer of the lower part of the motor region.

G. V. N. DEARBORN.

#### PATHOLOGY.

- 117 LES ARTHROPATHIES TABÉTIQUES ET LA RADIOGRAPHIE (The Tabetic Arthropathies and Radiography). Gibert (Nouvelle Iconographie de la Salpêtrière, 13, 1900, p. 145).

A paper having for its purpose, by means of radiographs, the explanation of certain anatomical details of tabetic arthropathies. The observations were made upon four cases: 1. An osteo-arthritis of the right thigh and exostosis of the right femur. 2. Osteo-arthritis of the right knee. 3. Arthritis of the femur, with involvement of the hip joint. 4. Osteo-arthritis of the left knee. The radiographs are included in the article. The following facts are noted as the result of these observations: The chief anatomical fact to be considered is the disappearance or the deformation of the contiguous articular surfaces, together with an hyperplasia of the adjoining osseous parts. The deformation is caused sometimes by the flattening of the normally rounded head; at other times by the disappearance of the articular cavity; sometimes the articulation disappears in the midst of the new formation; at other times the articular ends preserve their morphological integrity and the new bone formation is formed round about the articulations. Osteophytes may be produced far from the joint involved, often upon the neighboring bone. Lastly, there is another mode of formation which has not yet been described. It is not by a progressive and continuous increase of the epiphyses, but by the formation of osteophytes in the periarticular surfaces. We are further justified in believing that a trophic form of tabes exists, and that there is in the cord a trophic center. The exact location of this is at present unknown. The excitation of this center has as the result an osseous hyperplasia, whereas a cause acting in an inverse



way results in an osteo-articular atrophy. Owing to the relation which seems to exist between sensory and trophic symptoms, as was first brought out by Grasset, there is reason to suppose that this center is to be found in the neighborhood of the sensory center, somewhere in the medullary territory. SCHWAB.

- 118 SARCOM DES III VENTRIKELS MIT METASTASEN IM IV VENTRIKEL (Sarcoma of the Third Ventricle, with Metastases in the Fourth). E. Meyer (Archiv. für Psych., XXXII, 1899, p. 320).

The clinical symptoms were cephalalgia, vomiting, optic neuritis. The psychical condition resembled that described by Korsakoff. The autopsy showed a tumor of sarcomatous nature filling the third ventricle, which was not adherent to the walls. In the fourth ventricle there were found two small masses the size of a pea of the same microscopical character as the tumor in the third ventricle. Meyer believes that these were metastatic growths following the course of the cerebro-spinal fluids. JELLIFFE.

- 119 ANÉURYSME DE L'ARTÈRE VERTÉBRALE GAUCHE (Aneurism of the Left Vertebral Artery). Autopsy. P. Ladame and C. von Monakow. (Nouvelle Iconographie de la Salpêtrière, January and February, 1900, 13th Year, No. 1).

Most cases of aneurysms at the base of the brain have no clinical significance whatever. They are generally accidentally discovered post-mortem, and have often merely a medico-legal interest. A short résumé of the case to which this article relates is as follows: A man, 68 years old, syphilitically infected in his thirtieth year; the present disease began to manifest itself in his sixtieth year by violent attacks of vertigo and of angina pectoris. Two years afterwards an apoplectic seizure followed an attack of vertigo more severe than any he had yet experienced. As the result of this his gait became distinctly cerebellar in character. Shortly after his speech became dysarthritic and he had attacks of dyspnea. The cerebellar ataxia was present on both sides, possibly a little more pronounced on the right. Death followed in a condition of stupor with Cheyne-Stokes breathing; towards the end he had difficulty in deglutition. Autopsy: The basal arteries were very tortuous; at the juncture of the basilar and the vertebral arteries on the left side an aneurysm about the size of a pigeon's egg was found. The structures around the aneurysm showed evidence of compression. The sixth pair of nerves was the only one which showed an effect from the aneurysm in the sense of a pressure atrophy. Some of the important facts brought out by the microscopic examination are the following: Medulla: The left pyramid was markedly atrophied in its entire course, showed a great decrease in volume, and presented a typical picture of compression atrophy. The left olive was also atrophied, both in the fibers surrounding it and in the nerve cells comprising it. The right corpus restiforme showed considerably atrophy. This was due possibly to the necrotic condition of the compressed left olivary body. An ascending degeneration similar to this has not as yet been described. The study of sections of the medulla and cord lower down showed changes on the left side which at times had the character of pressure atrophy, at times of secondary degeneration. The hypoglossus nucleus on both sides was found to be normal. The nuclei of the cranial nerves were for the most part normal, with the exception of a slight asymmetry in the two sides of the descending root of the trigeminal and of the left acoustic tubercle. The corpora restiformia presented singular appearances, in addition to

the atrophy of the right already spoken of. The left corpus is atrophied as the result of direct pressure by the aneurysm. Especially is this so in its caudal portion. The greater part of the left corpus restiforme had undergone a secondary degeneration as the result of the necrosis in the left cerebellar hemisphere. The changes in the cerebellum were most pronounced in the left hemisphere and peduncle. The pathologic changes of this case are described in great detail, the more important ones have only been mentioned in this abstract. The pathogenesis of these aneurysms of the vertebral artery are far from being understood. In this case the modification found in the medulla, the cerebellum, and the suboptical region are particularly important. The literature of the subject is carefully considered in the article, and the plates accompanying it add much to the understanding of the anatomical description.

SCHWAB.

### THERAPY.

- 120 TRAITEMENT DE L'EPILEPSIE (Treatment of Epilepsy). Rommé (La Presse médicale, Mar. 21, 1900).

The author has followed de Fleury's method of caring for epileptics for eight years. The arterial pressure is regularly observed and measured, and urinary toxicity tested at stated intervals. The epileptic, being a depressed individual, is subject to dyspepsia and sluggish nutrition, therefore whatever affects arterial pressure may precipitate an attack. Combined bromides lower excitability of the cerebral cortex. Nocturnal cases should be given a single large dose at bedtime. Hydrotherapy, salt baths, massage, etc., with the injection of small quantities of normal salt-solution at various intervals, have been found of great benefit. No water is allowed with meals and the diet should seek to prevent the possibility of gastro-intestinal fermentation. To eliminate toxic substances, moderate exercise, purgatives, diuretics, and diaphoretics, should be employed. The author recommends quiet life in country villages for the epileptic.

CLARK.

- 121 TREPHINING FOR TRAUMATIC EPILEPSY. Lamboth (La Presse med. Belges, Jan. 28, 1900).

Lamboth relates the following case: Youth of twenty-one years of age had severe epileptic convulsions for eight years. Thickening of left parietal, surface size of palm, developed toward cranium and much lessening its cavity. Trauma originated in a fracture *in situ*, possible separation of two tables having been followed by proliferation of diploë, or perhaps more probably an infectious diploëitis had occurred; ophthalmoscopic evidences of compressions absent. Patient had fallen from a height and struck his head; had been a typical epileptic for thirteen years, but development and intelligence had not suffered. Craniectomy was performed and a piece of bone 10 Cc. by 3 Cc. removed. It is stated that his attacks became much changed in type and immediately after the operation occurred at night only.

CLARK.

- 122 HEMI-CRANIECTOMY FOR EPILEPSY. Lamprasi (Annali di nevrolgia, 1900, XVII, fasc. VI, p. 414).

Lamprasi describes the following case: A youth, aged twenty years; epileptic. Left parieto-frontal region much depressed; attacks of convulsions typical, but without aura or monospasm; no mention of trauma. It was decided to remove one-half of the skull, and a horse-shoe incision was made from front of ear to rear of same; an osseous pedicle was left. Forty days after the operation patient had continued free from seizures.

CLARK.

## Book Reviews.

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LES LÉSIONS DU SYSTÈME GRAND SYMPATHIQUE DANS LE TABES, ET LEUR RAPPORT AVEC LES TROUBLES DE LA SENSIBILITÉ VISCÉRALE. Par le Dr. Jean-Ch. Roux. Georges Carré et C. Naud, Paris, 1900.

The French theses are often valuable additions to medical knowledge. The most recent of a brilliant series issued from the service of Prof. Dejerine is by Dr. J. C. Roux, and is on a subject of much importance, viz.: The condition of the sympathetic system in tabes, and the symptoms depending on the alteration of this system. Dr. Roux's thesis is less bulky than some that are published, but it contains the results of thorough and original work, and one reads it with a feeling of satisfaction, because he is not obliged to skip pages filled with well-known facts presented as though the author believed that in a multitude of words there is wisdom.

Comparatively little study has been given to the condition of the sympathetic system in tabes, and Dr. Roux has chosen almost an untilled field. He has studied in seven cases of tabes the large splanchnic nerves and the cervical and thoracic portions of the sympathetic chain. The fibers of Remak, the non-medullated fibers, are difficult to differentiate and their alteration escapes detection; Dr. Roux therefore has examined only the medullated fibers. The small medullated fibers are far more numerous in the sympathetic system than are the large ones, and the former Dr. Roux has found degenerate in tabes. He has made laborious counts of the nerve fibers of the sympathetic system, and has found that about one-half of the normal number of small medullated fibers disappear in tabes, while the large medullated fibers remain unaltered. He was unable to find alteration of the nerve cells in the sympathetic ganglia examined by him. Graupner likewise could not find alteration of these cells in a case of combined sclerosis, although he found degeneration of the nerve fibers of the sympathetic system. Dr. Roux has attempted to determine the origin of the small and large medullated fibers in the sympathetic system, and has experimented on the cat. Division of the anterior and posterior spinal roots caused degeneration of a considerable number of the small medullated fibers, but the large ones remained intact; if, however, the spinal ganglia were removed many of the large fibers degenerated. From these experiments he concludes that the latter fibers arise in the spinal ganglia and have a sensory function, while the small medullated fibers come from the spinal cord through the anterior and posterior roots. The normal condition of the large fibers in tabes would seem to indicate that the spinal ganglia are not diseased. The disappearance of many of the small medullated fibers in the sympathetic system was the result of degeneration of the posterior spinal roots, and Dr. Roux has found when he examined the posterior roots of the lower cervical and upper thoracic region in tabes that the small nerve fibers in these roots had almost entirely disappeared.

The clinical portion of Dr. Roux's work, and the application of his findings in explanation of the symptoms of tabes, are especially interesting and valuable. It is certain, he thinks, that the small medullated

fibers of the sympathetic system in the posterior spinal roots, except perhaps in the lumbar region, have a sensory function; the motor fibers of the sympathetic come from the vagus or the anterior spinal roots. The disturbance of visceral sensibility in tabes is therefore the result of destruction of sympathetic fibers in the posterior spinal roots. The anesthesia of the testicle, of the bladder, of the mammary gland and of the trachea, *i. e.*, absence of the peculiar discomfort caused by pressure over the trachea below the cricoid cartilage, are symptoms of disease of the sympathetic system. Ocular symptoms in tabes, including the Argyll-Robertson phenomenon, are not the result of the degeneration of the nerve fibers in the cervical sympathetic, because ocular symptoms from disease of the sympathetic system are the result of involvement of the anterior roots in the cervico-thoracic region, and the alteration in the cervical sympathetic chain in tabes is dependent on degeneration of posterior root fibers. Dr. Roux has found the sympathetic fibers normal in a case of paretic dementia in which Argyll-Robertson's phenomenon existed, and from this he concludes that this sign is not of sympathetic origin.

The discomfort produced by a blow over the epigastric region may not be experienced if the patient is tabetic, and this loss of sensation is the result of alteration of the solar plexus and splanchnic nerves.

The theory of a syphilitic origin of tabes has led to the use of iodide of potassium in the treatment of the disease. Dr. Roux shows that this medication may have serious consequences. The iodide of potassium may irritate the stomach, alter the mucosa, and cause more or less intense gastritis. He has seen symptoms of dyspepsia develop in about a dozen cases of tabes after the administration of the iodide. Pain is experienced in the lower portion of the thorax, especially in the left side, and may be bilateral, and sometimes is very severe. It is felt about an hour or two after eating, increases gradually in intensity, and then disappears to return when the next repast is taken. It is increased by anything which causes gastric irritation, and may be associated with vomiting, by which it may be relieved. The omission of the iodide and restriction to a milk diet are usually sufficient to overcome the pain in the side. The pain produced by the administration of the iodide may finally cause typical gastric crises, as may dyspepsia from other causes in tabetic patients, but these crises differ from the true tabetic gastric crises in their gradual onset and gradual disappearance, in their development after gastric irritation, or at the menses, and in their suppression when a milk diet is taken and the use of the iodide is discontinued. All this is knowledge of very practical importance.

SPILLER.

THE  
Journal  
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Nervous and Mental Disease.

Original Articles.

STUDIES IN ASTEREOGNOSIS.

SUMMARY OF THE RESULTS OBTAINED IN ONE HUNDRED AND FOURTEEN MISCELLANEOUS CASES OF NERVOUS DISEASE.\*

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The subject of astereognosis<sup>1</sup> is one of increasing importance and one which has not as yet received the attention which it merits. Many years ago B. Puchelt,<sup>2</sup> in 1844, directed attention to several cases in which the patient was unable to recognize foreign bodies by touch, although cutaneous sensibility was more or less preserved. Long after, Hoffmann,<sup>3</sup> in 1883, again took up the subject. He studied sixteen cases, in a majority of which the symptom was present. More re-

\*Presented at the twenty-sixth annual meeting of the American Neurological Association, May 1, 2, and 3, 1900.

<sup>1</sup>The word stereognostic was invented by Hoffmann and first employed by him in "Stereognostische Versuche." It is derived from *στέρεος*, solid, and *γινώσκω*, to know. The words stereognosis and astereognosis were, I believe, first used by myself in the presentation of the case of tumor of the oblongata. Stereognosis would, of course, mean the faculty of being able to recognize foreign bodies and astereognosis would imply the absence or loss of this faculty.

<sup>2</sup>Medicinishe Analen, X Bd., IV Heft, Heidelberg, 1844, p. 485.

<sup>3</sup>Herman Hoffmann, "Stereognostische Versuche," Inaug. Dissertation, Strassburg, Kontanz, 1883.

cently Wernicke,<sup>4</sup> Monokow,<sup>5</sup> Burr,<sup>6</sup> Dubbers,<sup>7</sup> Olmstead,<sup>8</sup> Sailer,<sup>9</sup> Williamson,<sup>10</sup> Vorster,<sup>11</sup> and Dejerine and Egger,<sup>12</sup> have described cases in which the same symptom was present. It occurred to the writer that it would be of value to carefully examine a large number of miscellaneous cases of diseases of the nervous system with reference to this symptom. In most of the cases thus far reported a lesion of the cerebrum was either definitely determined or the symptoms pointed to the cerebrum as the seat of the disease. At the last meeting of the American Neurological Association the writer reported a case of tumor of the oblongata<sup>13</sup> in which astereognosis was present, and it was especially the findings in this case which suggested a study of the subject in the hope that some conclusions, first as to the factors which enter into astereognosis, and secondly as to the clinical value of the symptom, might be reached.

Hoffmann pointed out that a large number of factors entered into stereognostic perception. These factors he separated into the tactile sense, the sense of location of tactile impressions, the sense or faculty of distinguishing two simultaneous impressions,<sup>14</sup> the pressure sense, the muscular sense, the temperature sense, the pain sense, the sense of knowledge of the position of the fingers to each other and to the hand, the recognition of passive movements and the recognition of active movements. The writer found, that for practical purposes it was best to separate the examination into the tactile sense, the

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<sup>4</sup>"Arbeiten aus der psychiatrischen Klinik in Breslau," Leipzig, 1895; also Monokow, "Gehirnpathologie," Nothnagel, Wien, 1897, p. 409.

<sup>5</sup>Monokow, "Gehirnpathologie," Nothnagel, Wien, 1897, pp. 410 and 411.

<sup>6</sup>Burr, JOURNAL OF NERVOUS AND MENTAL DISEASE, Jan., 1898.

<sup>7</sup>Dubbers, Neurologisches Centralblatt, 1897, p. 61.

<sup>8</sup>Olmstead, JOURNAL OF NERVOUS AND MENTAL DISEASE, Nov., 1898.

<sup>9</sup>Sailer, JOURNAL OF NERVOUS AND MENTAL DISEASE, March, 1899, p. 161.

<sup>10</sup>Williamson, British Medical Journal, 1897, II, p. 787.

<sup>11</sup>Vorster, Archiv f. Psychiatrie, Bd. XXX, No. 2, 1898, p. 341.

<sup>12</sup>Dejerine et Egger, Revue Neurologique, 1899, p. 891.

<sup>13</sup>Dercum, JOURNAL OF NERVOUS AND MENTAL DISEASE, August, 1899.

<sup>14</sup>"Raumsinn or space sense."

temperature sense, the pain sense, the pressure sense, the sense of weight or muscular sense, the sense of location (that is, the ability of the patient to correctly refer the impression to the point or area touched), the knowledge of the patient in regard to the position of the fingers, the condition of the fingers and hands as regards mobility, and lastly, but most important of all, the ability of the patient to distinguish one or more impressions made upon the hand and fingers at one and the same time, and to this faculty the writer proposes the name of the *spacing sense*. The faculty of perceiving objects by means of the hands is an exceedingly complex one, and an effort was made to determine if possible something as to the relative importance of the various factors here enumerated.

The objects used in making the tests were small cubes, oblong blocks of wood, marbles, balls, pebbles and common household and personal articles, such as spoons, thimbles, pen-knives, spools, scissors, nails, screws, etc. The patients' eyes were, of course, always bandaged during the tests.

In all the writer examined one hundred and fourteen miscellaneous cases in the nervous wards of the Philadelphia Hospital. Among the diseases studied were hemiplegia, diplegia, posterior sclerosis, ataxic paraplegia, myelitis, syringomyelia, multiple sclerosis, cerebro-spinal syphilis, multiple neuritis, epilepsy, paralysis agitans, alcoholism, chronic lead poisoning, senile tremor, traumatic neurosis and chronic rheumatoid arthritis. It was thought best, also, because of the large number of cases studied, not to present here detailed histories of cases, but merely a brief statement of the results obtained.

The investigation was limited to the ability of the patient to recognize objects by the hand. The hand is, of course, the principal organ of the stereognostic sense, although this faculty is not limited to the hand, but is shared to some extent by the soles of the feet, and to a smaller extent by the general surface of the body.

HEMIPLEGIA.—Beginning with hemiplegia, the number of cases examined was forty-nine. Of these eight were rejected because the mental condition either on account of the lesion or the age of the patient was such as to make an accurate study impossible. This reduces the actual number of

cases to forty-one. In examining cases of hemiplegia it should be stated the results obtained in the paralyzed hand were always compared with those obtained in the hand of the healthy side. In the forty-one cases it was rather surprising to find astereognosis present in twenty, *i. e.*, in almost one-half. In these twenty cases the astereognosis was found to be incomplete or partial in seven and complete or total in thirteen.

Studied in detail these thirteen cases revealed the following remarkable facts: The tactile sense was lost completely in only three. It was more or less preserved in ten; it was completely preserved in six and partially preserved in only four. In these ten cases the temperature sense was preserved in five and diminished in five; it was lost in none. The pressure sense was preserved in six, diminished in four and lost in none. The pain sense was preserved in five, diminished in five and lost in none. The spacing sense, on the other hand, revealed a very great degree of loss. Of the entire thirteen cases of total astereognosis, the spacing sense was lost in ten; it was preserved in one, diminished in one and not satisfactorily examined in one because of aphasia. These results are the more remarkable when we reflect that in these thirteen cases the tactile sense was lost in only three, while the other senses were, as we have just stated, preserved in varying degrees. In two only of these thirteen cases could the astereognosis be ascribed to a general anesthesia, *i. e.*, to a loss of all forms of sensation. In one case there was anesthesia to the tactile sense and to the temperature sense, but merely hypesthesia to the pressure sense, together with full preservation of the pain sense. The preservation of the pain sense and the pressure sense in this case was of no value in enabling the patient to perceive foreign bodies. In four of the thirteen cases all of the cutaneous senses, the tactile, the temperature, the pain and the pressure senses, were preserved, except the spacing sense. This was completely lost, and to this loss the astereognosis appeared to be due. In two others of these thirteen cases there was diminished tactile, temperature, pain and pressure senses, together with loss of the spacing sense. In another case there was hypesthesia to the tactile, temperature and pain senses, while the pressure sense was fully preserved, but here again the loss of the spacing sense gave rise to astereognosis.



In only one case, in which astereognosis existed, was the spacing sense preserved. In this case there was present in addition to the spacing sense a fully preserved tactile sense, a slightly diminished temperature sense, a preserved pressure sense and a very good power to estimate weight. There was, however, a decided loss in the ability to correctly refer the cutaneous impressions to the part touched, and a marked impairment of the power to describe the position of the fingers when the eyes were closed; in addition, there was present also a marked secondary contracture of the hands and fingers. Of the remaining two of this group of thirteen cases, one presented a marked hypesthesia of all the cutaneous senses together with marked secondary contracture, and the last a preservation of the various forms of cutaneous sensibility, but with loss of the power of location. The last mentioned case also suffered from a partial motor aphasia, and it was difficult to determine the condition of the spacing sense. The latter was, however, undoubtedly disordered. It is interesting in connection with this patient to state that he could readily distinguish objects with his left hand, which was the hand of his sound side. If when his eyes were bandaged an object were placed in his left hand and then placed among a number of other objects, the bandage having been removed, he could then correctly select the object which he had formerly held in his left hand. With the right hand this test invariably failed, proving the undoubted co-existence of astereognosis with the aphasia.

The various values of the sensory phenomena present in astereognosis as observed in these thirteen cases of hemiplegia can be gleaned from the following statements: The tactile sense was studied in twenty cases of complete and partial astereognosis; it was preserved in eleven, diminished in six, and lost in three. The temperature sense was studied in nineteen cases and was found preserved in ten, diminished in six and lost in three. The pressure sense was studied in eighteen cases and was found preserved in ten, diminished in six and lost in two. The pain sense was studied in eighteen cases and was found preserved in ten, diminished in five and lost in three. The sense of weight was studied in ten cases and found preserved in five, diminished in three and lost in two. The sense of

localization, *i. e.*, the power of correctly referring the impression to the part touched, was studied in six; it was found preserved in two and lost in four. The power to correctly describe the position of the fingers, the eyes being closed, was tested in nine and found preserved only once, diminished in six and lost in two. The spacing sense, the power to separate or distinguish multiple impressions, was studied in sixteen and was found preserved in only four, diminished in two and lost in ten. Very marked secondary contracture existed in ten cases. In one case there was present athetosis, but the value of this symptom in causing astereognosis is open to doubt when I add that in this case there was also loss of the spacing sense, all of the other cutaneous senses being preserved.

Secondary contracture of itself was found to be insufficient to determine astereognosis. In one case in which the contracture was excessive and in which there was present no voluntary motion of the fingers, all of the cutaneous sensibilities being preserved, together with the spacing sense, stereognosis was but slightly impaired; this patient, I should add, however, was an unusually bright woman. She could recognize a key or other complex body laid on the back of her hand or upon her fingers with great readiness. Further contracture even when associated with marked athetoid movements is not sufficient to determine astereognosis. In a young woman, aged twenty-eight, suffering from spastic infantile hemiplegia, there was present marked contracture of the left hand with marked and ceaseless athetosis. Notwithstanding, there was present complete stereognostic perception in this hand.

Loss of the sense of weight alone is insufficient to determine astereognosis. In a case of hemiplegia with marked secondary contracture but with full preservation of all of the cutaneous senses, there was present a loss of the sense of weight. The patient could accurately describe various articles placed in his hands, but every now and then would make gross errors in attempting to describe their weight. This would occasionally lead him to describe a piece of wood resting upon his hand as a piece of iron, but even then he would accurately describe its shape.

One of the most remarkable findings occurred in a case of

hysterical anesthesia. This patient was presented before the Section on Internal Medicine of the College of Physicians by my colleague, Dr. Burrr, but the case has, I believe, never been published by him. The history is briefly as follows: The patient was a woman, white, thirty years of age, who suffered from left-sided hemiplegia. The arm was merely paretic and its mobility varied at various times. There was no, or very slight, spasticity of the leg. The knee-jerks were slightly increased. There was no ankle-clonus. There was no contracture in either the arm or the leg. There was complete anesthesia in the entire left half of the trunk, the left arm and the left leg. The patient slightly dragged her left leg in walking and at times walked very much better than at others. The case was regarded as hysterical, and, being tested for the stereognostic sense, revealed the following remarkable condition: The tactile sense, the temperature sense, the pressure sense and the sense of weight and, of course, of necessity, the spacing sense, were lost. She also knew nothing of the position of the fingers. When, however, complex bodies, such as a key, scissors or a pen-knife, were placed in the paralyzed hand, she instantly recognized and correctly named the object. She was examined repeatedly and only made occasional errors. These arose, however, always in reference to the weight of bodies. For example, while she described accurately the shape of a piece of card-board, she declared that it was a piece of sheet iron. This error she made repeatedly. When we consider the fact that the left side was paretic, although the hemiplegia was hysterical, it is suggestive, to say the least, that the only factor of the stereognostic sense which was really lost, was the sense of weight. The preservation of the stereognostic sense in the hemianesthesia of hysteria is not altogether unknown, but it is certainly excessively rare. Hoffmann made a similar observation in hysterical anesthesia. (Case XV.)

It may be worth while to briefly restate my findings in the forty-one cases of hemiplegia. In twenty cases astereognosis, more or less pronounced, was present. In eight cases the patients suffered from right-sided hemiplegia and in twelve from left-sided hemiplegia. That this proportion has, however, no clinical value, is revealed by the fact that a similar proportion

was found between my cases of right and left hemiplegia in which no astereognosis existed.

Before dismissing the cases of hemiplegia, let me suggest that it would be most interesting to study astereognosis in cases of aphasia. It would be very difficult, but the method by which it may at least occasionally be accomplished, I have already indicated.

**DIPLEGIA.**—Five cases of diplegia of childhood were studied. In two the stereognostic sense was markedly defective, apparently because of excessive athetoid movements. In another, while marked athetosis and contracture existed upon both sides, astereognosis was found only upon one, and an examination of this side revealed a complete hemianesthesia, a total loss of all forms of sensation. In a third case of diplegia, due to internal hydrocephalus, astereognosis existed in the right hand only. In this case the various cutaneous senses were preserved, but to what extent the patient's intelligence did not permit me to determine, and it was unfortunately not possible to determine the cause of the astereognosis. Probably it was purely cortical in origin. In the remaining two cases of diplegia, there was no stereognostic loss whatever.

**LOCOMOTOR ATAXIA.**—Sixteen cases of locomotor ataxia were studied. Astereognosis existed in five cases and in both right and left hands. It was total only in three and partial in two. As in the cases of hemiplegia, the hands alone were studied. In three cases in which complete stereognostic loss existed, the tactile sense was preserved in one and diminished and retarded in two. The temperature sense was preserved in all. The pressure sense was diminished in all. The pain sense was preserved, but slightly diminished in all. The spacing sense was much diminished in two and entirely lost in one. The sense of weight was much diminished in one, slightly diminished in one and lost in the third. The sense of location was preserved or but slightly diminished in all; it was not lost in any. The ability to recognize the position of the fingers was lost or greatly diminished in all three. Marked ataxia of the hands and arms existed in all three. In two cases in which the astereognostic loss was pronounced, but not complete, the tactile sense was preserved in both with retardation in one.

The temperature sense was preserved in both. The pressure sense was preserved in one and diminished in the other. The pain sense was preserved in both. The spacing sense was much diminished in both. The sense of weight was diminished in one and lost in the other. The sense of localization of impressions was preserved or but slightly diminished in both. The ability to recognize the position of the fingers was diminished in both. Both cases presented marked ataxia of the hands and arms. In the eleven cases of locomotor ataxia in which there was no stereognostic loss, the examination of the various forms of sensation in the hands yielded negative or inconclusive results.

In studying the findings in the cases in which astereognosis existed, we are impressed with the fact that as in the astereognosis of hemiplegia, the spacing sense was much involved; though not lost, it was diminished in all five cases. It is very probable, however, that the ataxia and the diminution of the knowledge of the position of the fingers were also of great importance in determining the astereognosis.

**ATAXIC PARAPLEGIA.**—Three cases of ataxic paraplegia were studied. Astereognosis was present in two; absent in one. In the two cases in which astereognosis existed, the tactile, temperature, pain and the pressure senses were preserved in both. The spacing sense was lost in both. The sense of weight was diminished or imperfectly preserved in both. The sense of localization was preserved in both. The knowledge of the position of the fingers was much diminished in one and lost in the other. Marked ataxia of the hands and arms existed in both. Here again we are impressed by the loss of the spacing sense, although the diminution of the knowledge of the position of the fingers as well as the ataxia must as in tabes be of importance.

**MULTIPLE NEURITIS.**—Two cases of multiple neuritis were studied. In one astereognosis was present in both hands. In the other no astereognosis existed. In the case in which the astereognostic sense was lost, both hands were studied as follows: In the right hand, the tactile sense was diminished and retarded. The temperature sense was preserved. The pressure sense was lost. The pain sense was

hyperacute. The spacing sense was lost. The sense of weight was lost. The sense of location of impressions, strange to say, was preserved. The knowledge of the position of the fingers was lost. In the left hand the tactile sense was preserved, as were also the temperature and pressure senses. The pain sense, as in the right hand, was hyperacute. The spacing sense was greatly diminished. The sense of weight was lost. The sense of localization, the pressure sense and the knowledge of the position of the fingers, as in the right hand, was lost. Here again the loss or great diminution of the spacing sense is the striking feature, although the absence of the power to appreciate the position of the fingers must also be regarded as of importance.

In all the other cases of nervous disease which were studied, eighty-three in number, no astereognosis was discovered. Thus, astereognosis was absent in five cases of myelitis, in five cases of multiple sclerosis, in three cases of cerebro-spinal syphilis, in one case of syringomyelia, in five cases of paralysis agitans, in eleven cases of epilepsy and also in the various other affections enumerated at the beginning of this paper.

CONCLUSION.—The above studies appear to justify the following conclusions:

First—It would appear that the loss or impairment of the spacing sense is the most important factor in determining astereognosis. It must be remembered, however, that astereognosis may exist though the spacing sense be preserved. In the single case of hemiplegia in which this was the case there was, however, a loss of location, a loss of the knowledge of the position of the fingers and marked secondary contracture.

Second—Next in importance to the spacing sense appears to be the knowledge of the position of the fingers and ataxia of movement.

Third—The mere preservation of the ability to perceive tactile impressions and the preservation of the pressure, temperature and pain senses is insufficient to prevent stereognostic loss.

The question next arises, is it possible to draw any conclusions of clinical value? It is evident, of course, that astereognosis may be due to lesions of the brain, of the medulla (au-

thor's case), of the cord, and of the peripheral nerves. Further, astereognosis must be of two kinds, peripheral or central; that is, it must be due either to an interruption of the pathways, the peripheral nerves or cord, by means of which the various sensory impressions reach the brain; or these pathways being open, it must be due to some lesion of the cortex. In many cases astereognosis is undoubtedly cortical. This is undeniably the case in dementia; it was also the case in a brain tumor involving the cortex, reported by Drs. Mills and Keen at this meeting. Now if astereognosis be at times cortical and at times peripheral, are there any means by which the symptom can be made of differential value?

Our present knowledge forbids a positive answer. It is not, however, impossible that it may be so employed. The various impressions received by the cortex from all of the various sources concerned in stereognostic perception must be combined in the cortex to give rise to the mental picture of the object felt. Given a case in which the various factors known to be important to stereognostic perception are all preserved and notwithstanding astereognosis exists, it would be justifiable to infer that the origin of the symptom is cortical. Again if with astereognosis there be specific or isolated losses, such, for instance as a loss of the sense of weight, or of the position of the fingers, or ataxia of movement without the signs of peripheral nerve or cord disease, it would, other things equal, justify the inference of brain or cortical involvement. Indeed, such a loss would indicate not merely cortical involvement; it would point directly to a special area of the cortex, namely, the posterior portion of the superior parietal lobule.

As to how much loss of the spacing sense depends upon nerve, cord or brain involvement, it is impossible to say. It appears to be present in astereognosis dependent upon lesions in any of these situations, and yet the function implied by the spacing sense is probably purely cortical. If we analyze the cases of locomotor ataxia and multiple neuritis in which the spacing sense is lost, we find that the tactile sense is generally either impaired or retarded, so that in them the loss of the spacing sense may be merely the expression of a tactile hyperesthesia.

## TWO CASES OF TUMOR OF THE SPINAL CORD.<sup>1</sup>

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The two cases of tumor of the spinal cord which are here presented in this paper differ considerably in course and duration, and especially in their anatomical character. The first case was one of endothelioma of the dura of the cord.

Case 1. The patient entered the Home for Incurables, on April 16, 1885, and was under the care of Dr. J. W. Courtney, whom I have to thank for permission to use the notes of the case. At the date of entrance the records of the hospital say that the patient, a single woman, twenty-two years of age, was born in Charlestown. Her father died of lead poisoning, and her mother of phthisis. She had had the ordinary diseases of childhood, and since she was seventeen years of age had done the work of a domestic servant. In June, 1884, she began to lose the use of the legs. In October of that year she could still walk with assistance, but at the time of her entrance to the hospital she had absolutely no power in the legs, while the arms were unaffected. Her temperature upon admission was 99.0° F. and her pulse 100. There was some constipation and incontinence of urine. The note says, "She is very comfortable." From the first of May to September of that year there was more or less pain during micturition, and also in the abdomen. The note in September, 1887, is that the patient was suffering from symptoms of cystitis and urethritis, and that she complained mostly of violent spasmodic and sudden contractions in her limbs. In November it is noted that there were beginning flexor contractures in the legs, that the patient was much emaciated, and that her mind was somewhat affected. In the following May it is noted that the patient's mental upset had culminated in an acute maniacal outburst. She broke all mechanical restraint, tore out her finger and toe nails, and attempted several times to strangle herself. She was sent to the McLean Hospital for the Insane, and later returned to the Home for Incurables. From December

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<sup>1</sup>Read at the twenty-sixth annual meeting of the American Neurological Association at Washington, D. C., May 1-3, 1900.



3 to April 1, 1888, there was decubitus of the right nates, and also on the left, which slowly improved. August 1, 1892, the note says, "Have straightened legs at most every visit, using all the strength I possessed, causing no pain, as she has no sensation in the legs." In April, 1893, it is noted that the patient was more hysterical than she had been in a long time, and not only depressed, but excitable, and that there was constant incontinence of urine. From this time on there are no entries in the records, but Dr. Courtney states that the patient was bedridden and extremely anemic. The cranial nerves were not affected, and although the pupils were pretty constantly dilated, they reacted to light, and upon accommodating. The upper extremities were free and the lower ones contracted and atrophied to a marked degree. The reflexes were not obtainable on account of the contractures. Sensibility in all forms was lost to a level on the trunk at about the mid-dorsal region. The function of the vesical and rectal sphincters was completely abolished. Decubitus was present. Exitus from exhaustion occurred on April 19, 1898. Autopsy by Dr. R. M. Pearce, on April 19, 1898. Body of woman about thirty-six years old, poorly nourished, and poorly developed. Decubitus over sacrum and right trochanter. Contractures of both lower limbs. Abdomen showed nothing abnormal on inspection. Pleural cavities normal. Pericardium normal. Valves and cavities of heart normal. Lungs normal. Spleen dark red in color and firm. Capsule smooth. On section; dark red in color and the Malpighian bodies not visible. Pulp not increased. Gastro-intestinal tract normal. Liver dark red in color and capsule smooth. On section, markings indistinct, and substance firm. Gall bladder normal. Kidneys irregular and nodular in shape; capsule peels leaving a rough, granular, uneven surface. In places there are rather deep depressions, which, however, do not appear to be scarred areas. On section the cortex is somewhat diminished, and the glomeruli appear as opaque points. Substance firm. Adrenals normal. Aorta normal. The bladder contains a considerable amount of whitish opaque fluid. Uterus, tubes and ovaries normal. Brain with its membranes normal. Middle ear normal. Spinal column. In the middorsal region of the vertebral column there is a projecting knuckle formed by the spinous processes of two vertebrae. Upon removing the cord there is found below the cervical enlargement, and at the level of the spine of the scapula, a granular reddish swelling of the dura, about 2.5 cm. in length. This tumor appears to be meningeal, but has almost completely replaced the cord beneath it. Below this point the

cord is considerably atrophied. There is no lesion of the cord opposite the vertebral knuckle.

Microscopic examination: The cord and tumor were hardened in formalin. Sections were made from the tumor, and at various levels of the cord, and stained with hematoxylin and eosin; methylene blue and eosin; and with Weigert's stain for myelin sheaths. Sections from the tumor about the cord show a tissue which varies considerably in various parts. In general it is composed of a mass of cells with large oval vesicular nuclei, rather closely packed together, with unstained cell protoplasm, and with no regular arrangement. In this mass of cells are numerous round bodies, made up of concentric rings, which stain homogeneously, but vary in color from a pinkish blue to a deep blue. About some of these round bodies can be seen two or three quite separate rings in which can be clearly made out the elongated nuclei of endothelial cells, such as are seen in the walls of blood vessels. Many of these corpora amylacea have been calcified. In places the tumor is made up chiefly of these bodies, with more or less connective tissue, and only in places an irregular mass of cells with large nuclei spoken of above. Here and there in the tumor are blood vessels, which appear normal. These are usually small. In a section taken from a slightly lower level, the relations of the tumor can be made out more exactly. It lies in the dura, and at this point only partially surrounds the cord in a concentric mass. The dura is much thickened by increase of connective tissue, and is divided by the tumor mass, which is surrounded by the thickened dura, as by a capsule. At this level, in addition to the calcified corpora amylacea, there are in the tumor mass itself, at its border near the thickened, surrounding dura, three small masses of bone, which show indistinct lamination, and lacunæ, which are more rounded than those seen in normal bone. Occasionally canaliculi can be made out running a short distance from the lacunæ. The bone contains a few marrow spaces in which are blood vessels, and a slight amount of loose, edematous connective tissue. At this level the pia is much thickened by increase of connective tissue. Some of the blood vessels of the pia show marked thickening of the media. The dorsal and ventral nerve roots at this level appear practically normal. The cord at this level shows only a few scattered fibers at the periphery which have retained their myelin sheaths. The cord in general at this level shows no signs of the normal structure. It is made up of a very dense meshwork of neuroglia fibers, which become less dense at the periphery, and in the gray matter of the cord.

Rarely a nerve cell can be made out, but those that are seen are rounded or flask shaped and show no signs of processes, or at most but a single one, and show either no signs of nucleus and nucleolus, or indistinctly staining ones. The only remains of the normal nerve fibers of the cord are a few scattered fibers at the periphery, and a rather larger number of fibers, grouped together at the periphery of the posterior columns, which have retained their myelin sheaths. Elsewhere the cord is completely transformed by the growth of neuroglia fibers, though the shape of the gray matter can still be made out from the surrounding part of the cord because of its lesser density. The nerve roots appear fairly normal. Section of the cord a little above the point of greatest compression, stained by Weigert's method, show the cord considerably lessened in size, and a very marked degeneration of the median part of the posterior columns. In this area practically no nerve fibers can be seen which have retained the sheaths. There is also a diffuse, but well marked, degeneration in the direct cerebellar tracts, and at the periphery of the cord throughout the lateral border, and also along the anterior fissure. The atrophy is very marked in the direct cerebellar tracts and less so in the other portions, where some normal fibers are seen among the degenerated fibers. Immediately below the point of greatest compression, the cord being still small in size and shrunken, the sections stained by Weigert's method show the usual descending degenerations. The cord is small and there is a well-marked indentation just anterior to the point of exit of the posterior nerve roots. There is a very marked degeneration of the fibers of the lateral and anterior pyramidal tracts, and a moderate diffuse degeneration at the periphery of the cord in its anterior and lateral parts, and a very slight diffuse degeneration throughout the anterior ground bundle. The nerve roots are normal. The degeneration at the periphery of the cord is no longer seen at the lower part of the dorsal cord, though the degeneration of the motor tracts, both lateral and anterior, is well marked here, as also that of the lateral pyramidal tract in the lumbar cord, after the anterior tract has ended.

Anatomical Diagnosis: Decubitus; chronic diffuse nephritis; chronic cystitis; contractures of lower limbs; endothelioma of the dura, with calcification, and bone formation; compression of the cord with secondary atrophy and sclerosis of the cord; ascending degeneration of the posterior, and direct cerebellar tracts, and descending degeneration of the pyramidal tracts, with diffuse degeneration of the tract of Gowers' and anterior ground bundle, and at the periphery of

the cord for a short distance above and below the point of compression.

To resume, we have in this case to do with a young woman who gradually became paraplegic during the course of ten months. At the time of admission to the hospital the sensory and motor paralysis, and the paralysis of the sphincters seems to have been complete, and was certainly so at a later period, and from that time on she seems to have had no pain, an unusual thing in tumors affecting the spinal cord, where pain and tenderness of the spinal column are apt to be early and fairly constant symptoms. Gradually very marked contractures developed, and the patient remained in much the same condition for a period of thirteen years, except for an outbreak of mental trouble with excitement, from which she made a good recovery. Death finally came to end the scene, probably caused by exhaustion induced by the bed-sores, the diffuse nephritis, and the chronic cystitis. The autopsy showed these conditions, as well as a tumor of the dura which proved to be an endothelioma which had produced a practically complete destruction of the nervous elements of the cord at the point of greatest compression with increase of neuroglia tissue and the usual ascending and descending degeneration of the various tracts of the cord.

Schlesinger<sup>2</sup> states that endotheliomata of the cord, usually proceed from the dura, and are often multiple. They constitute one of the rarer forms of tumors affecting the spinal cord.

Mader<sup>3</sup> has recently reported a case which was fatal in twenty-nine days from the time that the symptoms were first noticed, and twenty-five days after entrance in the hospital. In this case there was found an endothelioma of the dura which the author thinks was affected by an acute swelling by which the rapidity of the disease was explained.

Orlowsky<sup>4</sup> in the Society of Neuropathologists and Alien-

<sup>2</sup>Schlesinger, H., "Beiträge für Klinik der Rückensmarks- und Wirbeltumoren." Jena., 1898, p. 37.

<sup>3</sup>Mader, J., "Perivaskuläres Sarcom der Dura Mater spinalis. Acute Schwellung derselben mit rapid sich entwickelnden Compressions-erscheinungen. Paraplegie. Grangraen. Cystitis. Tod." *Weiner med. Blätter*, 1898, XXI, 249.

<sup>4</sup>Orlowsky, *Neurolog. Centralbl.*, 1898, XVII, 92.

ists at Moscow, at the meeting October 22, 1897, reported a case in a fourteen year old girl, where the disease began with mild pain in the back, and progressive weakness of the legs. Three months after the beginning of the symptoms she was admitted to the hospital, and at that time there was complete paralysis of the legs, with flaccid muscles, and atrophy, absent knee-jerks, lively Achilles tendon reflex, anesthesia over the whole of the lower extremities, except the posterior surface of the thighs, and the outer side of the lower legs, to a point four fingers breadth above the navel in front, and to the level of the ninth dorsal spine behind, with a slightly hyperesthetic zone above this anesthetic one, and with retention of urine and feces. In the course of the disease the anesthesia increased, paresthesiæ and intention tremor of the hands came on, followed by anesthesia of the ulnar region of the arms, and paresis of the arms, with marked wasting of the legs. The patient died fourteen months after the onset from septicemia and disturbance of respiration. The autopsy showed an endothelioma of the pia, which had destroyed the cord. The growth was vascular, and contained obliterated, and hyaline degenerated, and calcified vessels. It also showed much connective tissue, which was also hyaline. In addition there was a gliosis in the posterior columns, with cavities, which were not lined with epithelium, and were not connected with the central canal, nor with the tumor. There was marked softening of the medulla, and some small metastases of sarcoma in the pia of the brain and cerebellum. Orlowsky ascribes the syringomyelia and gliosis to stasis produced by the tumor, and thinks the case favorable to the theory of Langhans and Kronthal of the origin of syringomyelia from stasis, but thinks that the central canal need not be involved, as they thought necessary.

Wersiloff<sup>5</sup>, at another meeting of the same society, on January 30, 1898, reported a case in which the duration of the disease was three and a half months. It began rather suddenly with symptoms of a spastic spinal paralysis of the left extremities, and diminished sensation on the right side

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<sup>5</sup> Wersiloff, *Neurolog. Centralbl.*, 1898, XVII, 563.

of the body and the right lower extremity. A few weeks later there was a complete paralysis of the right extremities with increased tendon and skin reflexes. Throughout the course of the disease there was no sign of compression of the posterior nerve roots. The autopsy showed a somewhat similar condition to that found in the case we are considering. The tumor was an angio-sarcoma psammomatousum, arising from the dura at the level of the second cervical segment, and compressed the cord markedly from the left side. Lancereaux<sup>6</sup> published a case of a woman who gradually became paraplegic with strong flexor contractures of the legs, loss of power over the sphincters, and considerable impairment, but not absolute loss of sensibility, where the course of the disease was extended over six years, and there was found a tumor growing from the inner surface of the dura, and compressing the cord in the mid-dorsal region, so that it was softened, and dark in color. The tumor, which was 3 cm. long, was a sarcoma in which the cells were arranged in concentric groups, the centers of which had undergone calcification.

Troitzky<sup>7</sup> reports a case of five years' duration, in which pain was the first symptom, and where there was a flaccid paralysis of the legs, with absent knee-jerk and tendo Achillis reflex, paralysis of the bladder and diminished sensation. The autopsy showed a tumor growing from the inner side of the dura at the level of the third lumbar segment, which was 3.5 cm. by 2.0 cm. in size. Microscopically it was composed of endothelial cells, arranged in bands and groups, in the center of which there could often be made out a lumen. There was no calcification. Clarke<sup>8</sup> reported a case in a man twenty-eight years of age. It began with weakness of the arms and legs, but only a dull aching sensation in the shoulders. Later there was paresis of the

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<sup>6</sup>Lancereaux, Quoted by Gowers, "Diseases of the Nervous System," Phil., 1892 2d ed., i., 545.

<sup>7</sup>Troitzky, S., "Ein Beitrag zur Kenntniss der Endotheliome der Pachymeninx spinalis." Prag. med. Woch., 1893, XVIII, 603.

<sup>8</sup>Clarke, J. M., "On endothelioma of the spinal dura mater; with a case in which an operation was performed." Brain, London, 1895, XVIII, 256.

legs and arms, with some wasting in the small muscles of the hand. The knee-jerks were increased, and there was ankle-clonus. The patient became completely paralyzed in ten days. Sensation was lost in the arms and body, but was present over the legs. The patient then left the hospital to return two years later desiring an operation. This was performed, and the tumor removed, but the patient died that night, apparently from shock, as he lost much blood during the operation. The tumor extended from the fifth cervical to the first dorsal segments. The author calls it an "endothelioma or psammoma, more properly the former, as it contained calcareous granules."

Drs. Alexandroff and Minor<sup>9</sup> at the meeting of the Society of Neuropathologists and Alienists at Moscow, on February 23, 1896, reported a case of compression of the cord at the level of the fourth and fifth cervical vertebræ, in which an operation was performed, but the patient died of pneumonia. At the autopsy an endothelioma of the vertebræ and dura was found.

Aside from these cases Schlesinger<sup>10</sup> speaks of one published by Eppinger,<sup>11</sup> in which the growth proceeded from the epithelioid cells of the subarachnoid space, and of another of Dionisi<sup>12</sup> proceeding from the dura. Schlesinger's own cases are those numbered 37 and 38.<sup>13</sup> The first was in a woman twenty-seven years of age. The first symptoms were pains, slight sensitiveness of the spine to pressure, with stiffness of the spine. One year later weakness of the legs appeared, and six months later still there was paraplegia with loss of the tendon reflexes, and with severe pains and bladder disturbances, and disturbances of sensation. The autopsy showed an endothelioma of the dura in the lumbar region, composed of spindle-cells, and with concretions similar to brain sand. In the second case Schlesinger gives no clinical

<sup>9</sup> Alexandroff and Minor, *Neurolog. Centralbl.*, 1896, XV, 1048.

<sup>10</sup> *loc. cit.*, p. 38.

<sup>11</sup> Eppinger, "Endothelioma der Pia spinalis." *Vierteljahresschrift für prakt. Heilkunde*, CXXV, 171.

<sup>12</sup> Dionisi, "Tumore del midollo spinale." *Società Lancisiana degli Opedali di Roma*, 1892.

<sup>13</sup> *loc. cit.*, p. 171-173.

history. At the autopsy there was found at the level of the third and fourth dorsal vertebræ a tumor springing from the arachnoid, which compressed the cord so that its continuity appeared completely broken. No details of the microscopical examination are given, but the tumor is called an endothelioma.

Dexler<sup>14</sup> reports two cases of tumors in dogs, the second of which was an extra-dural tumor, 2.5 cm. long, which microscopically was an endothelial sarcoma, and had produced compression of the cord with characteristic symptoms.

In the consideration of the endotheliomata of the cord, however, there seems to be considerable uncertainty whether psammomata are to be classed as a form of tumor separate from the endotheliomata or not. Schlesinger<sup>15</sup> says that a sharp differentiation of these tumors from the endotheliomata apparently cannot be drawn, and proceeds to describe the psammoma as composed of small spindle cells, which are fused into concentric circles, and says that the proper nature of these formations, which are characteristic of psammomata, is still in dispute, and that it is not certain whether they are an epithelial product or, as seems more probable, altered vessels. He then states that he has collected fourteen cases from the literature, and adds four from his own material. The one of Lancereaux, those of Blondel, Berger, and Oustaniol were inaccessible, but a study of the others, Schlesinger's own cases, numbers 39, 40, 41 and 42 in his series, in general reported very briefly, and Ernst's cases of psammoma of the cerebral meninges, seems to show that the psammoma as described have varied in character. In Wilks<sup>16</sup> case there is no description sufficient to determine the nature of the tumor, and Hutchinson,<sup>17</sup> though describing his case as a psammoma, says it strongly suggests an endothelioma, in

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<sup>14</sup>Dexler, H.; "Zwei Fälle von Tumoren der Rückensmarkshäute." Monatshefte für prakt. Thierheilkunde, VII. Ref. in Neurolog. Centralbl., 1896, XV, 852.

<sup>15</sup>*loc. cit.*, p. 38.

<sup>16</sup>Wilks, "Tumor of the spinal cord." Transactions Pathological Soc., London, 1855, VII, 37.

<sup>17</sup>Hutchinson, J. W., "Psammoma of the spinal cord." Transactions Pathological Soc., London, 1882, XXXIII, 23.



which partial calcification had occurred. Lediard's<sup>18</sup> case, classed by Schlesinger as a psammoma, he himself calls an endothelioma of the pia. Cayley<sup>19</sup> speaks of the concentric arrangement of the cells, and of calcification, and Whipham's<sup>20</sup> description is very similar. Steudener<sup>21</sup> gives a very insufficient description of the appearance of the tumor, but calls it a psammoma, as Pal<sup>22</sup> does his case. In the Verein für Wissenschaftliche Heilkunde at Königsberg, Lichtheim<sup>23</sup> reported two cases operated upon, one of which died of meningitis, while the other recovered, and could walk again, and in both instances he states that the tumor was a psammoma, but no description of the appearances was given. Bailey<sup>24</sup> also reports a case of psammoma of the dura, at the level of the seventh and eighth dorsal segments, which pressed upon the posterior columns, but had run its course without symptoms, except the loss of the knee-jerk, and without pain, the tumor being found at the autopsy. He reports in the same paper a case of syphilitic affection of the meninges, which also had produced no pain, and concludes that the presence or absence of pain, in cases of tumor affecting the spinal cord, is due more to the location of the tumor than to its particular variety. Ernst<sup>25</sup> in his paper certainly shows that these growths are not epithelial in origin or character, but probably proceed from the blood vessels, or lymph spaces, and are properly endothelial, and a study of the cases reported seems to show that while it is possible that under the psammomata

<sup>18</sup>Lediard, H. A., "Tumour of dura mater, pressing upon the cord in the cervical region." Transactions Pathological Soc., London, 1882, XXXIII, 25.

<sup>19</sup>Cayley, W., "Tumour pressing on the spinal cord." Transactions Pathological, Soc., London, 1865, XVI, 21.

<sup>20</sup>Whipham, T., "Tumor of the spinal dura mater, resembling psammoma, pressing upon the cord." Transactions Pathological Soc., London, 1873, XXIV, 15.

<sup>21</sup>Steudener, F., "Zur Kenntniss der Sandgeschwülste." Virch, Arch. f. path. Anat. u. Phys., 1870, I, 222.

<sup>22</sup>Pal, J., "Ein Fall von Rückensmarks-compression." Wiener klin. Woch., 1892, V, 350.

<sup>23</sup>Deutsche med. Woch., 1891, XVII, 1386.

<sup>24</sup>Bailey, P., "Report of two cases of tumor of the spinal cord, unaccompanied with severe pain." JOURNAL OF NERVOUS AND MENTAL DISEASE, 1896, XXIII, 171.

<sup>25</sup>Ernst, P., "Ueber Psammome." Beiträge z. path. Anat. u. z. allgem. Path., 1891-2, XI, 234.

there have been included some cases of sarcoma, or other growths with calcification, in the majority of cases we have to do with endotheliomata proceeding from the blood vessels, or lymph spaces, and varying in the amount of irregularly arranged cell masses, those where this has predominated having been usually classed among the endotheliomata, while cases in which the concentrically arranged cell masses were numerous, and particularly where there has been calcification, have been classed as psammomata. Gowers in the case of Lancereaux, referred to above as an endothelioma, calls it a sarcoma in which the cells were arranged in concentric groups, the centers of which had undergone calcification. It seems to me that these growths are certainly endothelial in origin, and cases, such as the one we have just presented, from the mixture of the tissues which apparently vary so much in structure, seem to show that these two classes of tumor are in reality but one. The presence of calcification in the tumor is not unusual, but the presence of true bone as in this case is much more rare, certainly in tumors, though more common following inflammatory processes. In this case we had, too, certain of the characteristics of psammomata, in the slow growth of the tumor and the long duration of the disease, with the marked compression of the cord, present for so long a time, without extension into the cord itself, and as in general such cases are well adapted for operative interference, we may feel that this case was one in which an early operation might have produced a brilliant result.

The second case I have to report occurred in a boy, six years of age, who came to the Department for Diseases of the Nervous System, at the Boston City Hospital, on May 31, 1899. His family and previous history threw no light upon the case. The account given at that time was that the symptoms were of two weeks' duration, and had followed a fall from a small cart while it was in motion, a distance of less than two feet, the boy striking first on the right elbow and then on the right shoulder and back. The next day he complained of pain in the elbow, and two days later the mother noticed that he could not open the hand to take a biscuit. Then he began to complain of intense pain in the right shoulder and neck, especially at night, and the mother noticed that the head was held fixed and not moved normally. The moth-

er also stated that while she was pregnant with this child she saw a man with an amputated left arm, which, at the time, made her feel very badly. When the child was born the left forearm was absent. Physical examination at the time of application showed a pale, not very well developed boy, with a congenital absence of the left forearm from a point about two inches below the elbow joint. The radius and ulna could be felt for a distance of about two inches. There was marked rigidity of the neck from spasm of the muscles, and the head was held inclined toward the right shoulder, with the chin rotated to the left and elevated. Passive movement of the chin to the left was possible to a slight extent, but not to the right. There was no marked tenderness of the spine to pressure, nor pain on pressure upon the head. There was weakness of the right arm and the fingers could be flexed and extended, but very slightly. Supination and pronation of the forearm was possible, but weak. The arm could be abducted to an angle of 120 degrees. There was no tenderness to pressure along the nerve trunks. Sensation was intact. The electrical reaction of the affected muscles was normal. The pupils were equal and reacted to light and upon accommodating. The heart was normal. The symptoms continued and the paresis of the right arm increased, so that it could not be moved, and on June 19 it was stated that he had been dragging the right foot in walking for about a week. Examination on that day showed the right knee-jerk increased, but no ankle-clonus. The strength was markedly diminished in the right leg, but the leg could be lifted from the bed and rotated and the foot inverted and everted; in fact, there seemed to be no definite paralysis, but only a diffuse paresis of this extremity. The head was held in the same position as at the first examination, and there was considerable spasm and rigidity of the muscles of the neck, but no deformity of the spine. Passive motion remained the same as before. The child was unable to move the right arm or hand, except that there was slight power of flexion of the forefinger. The right shoulder could be elevated. The right hand was somewhat swollen. There was very slight quantitative change in the reaction of the muscles of the right arm to the faradic current. The pupils were equal and reacted normally. Sensation was intact. The right cremasteric reflex was increased. The urine was negative. The same day the patient was admitted to the hospital on the service of Dr. Cushing, to whom I am indebted for permission to use the notes taken in the hospital. The patient was put to bed with an extension weight of five pounds. On June 24 it was noted that there was still some

muscular spasm, especially in the right scapuli. The power of the right arm remained the same, but the right leg was apparently normal. On June 26 it was noted that the right patellar reflex and the right cremasteric reflex were less than at entrance. On June 28 it is noted that the muscular spasm in the neck was diminishing. On July 1 the note is that the paralysis of the right arm continued and through all this time it is repeatedly stated that the child was comfortable, or "as comfortable as could be expected." The temperature during his stay in the hospital varied somewhat, usually above 99.0° F. and going as high as 100.2° F. The pulse varied between ninety and one hundred. On July 4 the note reads that the temperature the previous night was 101.4° F.; pulse 120; that he ate as well as usual, had no cough; that the glands on the right side of the neck were enlarged and there was marked swelling back of the left ear and above the base of the mastoid, which was apparently not tender. The examination of the chest was negative; no abnormal signs were found. About twenty or twenty-five minutes before death the child complained of severe pain in the back of the neck, which became steadily worse. The extension apparatus was removed and the pillow taken from under the child's head. He became cyanotic, respiratory movements of the chest being scarcely perceptible. The cyanosis increased and the child became unconscious, but without struggling. A loud systolic murmur was heard over the cardiac area, but the pulse was regular and full. Subcutaneous injections of ether and strychnia were given and artificial respiration was kept up for twenty minutes. There was discharge of considerable mucus from the mouth. The head was lowered and the feet raised. The pulse gradually failed and death which ensued was apparently due to failure of respiration.

Autopsy, July 4, 1899. Two and a half hours after death. Body 111 cm. long. Well built and fairly nourished. No decomposition and no marked lividity. Abdomen rather prominent. In the precordial region there is an area of cutaneous congestion, the size of a silver dollar. There is a congenital absence of the left forearm below the upper third. Pupils equal and moderately dilated. The peritoneal, pleural and pericardial cavities contain but the normal trace of fluid and their linings are normal. Diaphragm on the left side stands at the fifth rib and on the right side at the fourth interspace. Heart, weight 70 gms. Heart is normal save for patches of chronic pericarditis. Measurements: Tricuspid valve, 8 cm. Pulmonary valve, 4.5 cm. Mitral valve, 7 cm. Aortic valve, 4.5 cm. Left ventricle, 8 mm. Right ventricle, 4 mm. Lungs

weigh 230 gms. and are edematous, but otherwise normal. Spleen weight, 35 gms.; normal. Liver weight, 600 gms.; normal. Kidneys, combined weight, 150 gms. They are slightly congested and the glomeruli are a little prominent. Brain weight, 1,500 gms., normal in appearance. The spinal cord presents the chief lesion. Section of the cord in the first cervical segment discloses at this level a hemorrhage into the gray matter, with apparently but little extension into the white columns. The ventral and dorsal cornua are equally affected and of a dark brownish-red color and slightly lessened consistency. The normal markings are absent and the cord appears translucent and grayish in color. Section at the junction of the middle and lower thirds of the medulla appears normal. The hemorrhage extends down somewhat into the cervical enlargement. On opening the dura a large subarachnoid clot is seen extending from the level of the seventh cervical segment to that of the fifth thoracic segment. This clot is chiefly confined to the left dorso-lateral aspect of the cord and produces a distinct local bulging not adherent to the dura. The posterior root fibers are involved on both sides and the point of greatest bulging is in the region of the second thoracic segment.

After hardening in formalin, sections were made across the cord at frequent intervals and the following appearances were noted: The cord throughout the cervical portion is much and diffusely enlarged. Section at the first cervical segment shows the cord of a grayish, translucent color, while the gray matter is the seat of a hemorrhage, dark brown in color, which is fairly sharply limited to this area, extending apparently but very little, if any, into the white matter. At a slightly lower level, about the third cervical segment, the cord shows the same grayish, translucent appearance, but more especially in the region of the right dorsal horn, where the surface on section bulges decidedly above the level of the rest of the section. This gelatinous growth takes up the whole of the right portion of the cord throughout the next two or three lower segments, showing in places a brownish discoloration as if from hemorrhage, while occasionally small brownish areas are seen in the dorsal part of the cord. A little lower the center of the growth has a decidedly brownish color, the translucent grayish color being evident more especially at the borders. Still lower, at the lower part of the cervical enlargement, the hemorrhage is larger and occupies the center of the cord, extending dorsally and broadening, in a general way occupying the portions of the section which normally contain the gray substance and the posterior columns. There is also a consid-

erable sub-pial hemorrhage at this level, chiefly at the posterior part, involving both dorsal roots, which extends down a distance of about 2.0 cm. into the upper thoracic cord. The hemorrhage is about 3 mm. in thickness and lower down involves the left dorsal side more than the right. Here in the upper thoracic region there is quite a large hemorrhage into the gray matter of the cord 3 mm. in diameter, occupying the region about the central canal. This continues downward for about 2.0 cm., where it is again larger and involves the ventral and dorsal horns of the gray matter and the region about the central canal. This hemorrhage in the right dorsal horn extends downward a distance of about 2.0 cm. Below this the cord on section appears normal macroscopically.

Microscopical examination: Sections of the cord were made at various levels, and stained with hematoxylin and eosin; hematoxylin followed by picric acid, and acid fuchsin; by Weigert's method for myelin sheaths; and by Mallory's phosphotungstic acid-hematoxylin, and neuroglia stains. Sections from about the level of the third cervical segment show a marked thickening of the dura by increase of connective tissue. The pia shows no marked changes. The ventral and dorsal nerve roots show no changes. The substance of the cord shows almost no trace of the normal structures, but is composed of a cellular tissue, rich in blood vessels. This tissue is composed of cells, with oval vesicular rather small nuclei, with but little cell protoplasm, which show no regular arrangement. In places the nuclei are smaller and more homogeneous in appearance. No mitoses and no giant cells are to be found. Sections stained for neuroglia fibers show their presence throughout the new growth; in places they are quite numerous, passing in a fine network between the cells and over them, and in other places but few are seen. Here and there in various sections are small areas, made up entirely of a dense meshwork of neuroglia fibers, with almost no cell nuclei to be seen. Some of these areas of neuroglia meshwork lie about the blood vessels. The central canal of the cord can be made out in some of the sections of the cord, but in others no traces of it can be seen. Rarely a nerve cell, shrunken and homogeneous in appearance, can be made out. At the periphery are occasionally small areas, which contain myelin sheaths, which appear fairly normal. No differentiation between the white and gray matter of the cord can be made out, as the whole of the center of the cord is taken up by the tumor mass described. There are quite numerous small hemorrhages throughout the section. At a point lower down the section of the cord shows practically no trace of

tumor formation, but at the right side of the cord the pia is densely infiltrated with cells resembling lymphoid cells. These are closely packed together and show no definite arrangement. The infiltration extends into the cord at the point of exit of the dorsal nerve roots, which are seen, fairly normal in appearance, outside of the pia. This infiltration of the cord quickly diminishes, but there is a moderate increase of nuclei in the whole of the posterior horn upon this right side and also about the central canal. At the part of the cord where the infiltration extends into the pia there is a large hemorrhage, and another one, more diffuse, is seen near and posterior to the central canal, and extends back into the left dorsal horn. The cord for the rest appears fairly normal, except that upon the left side there are but very few nerve cells in the anterior horn, though those seen appear normal. These nerve cells are limited to the ventral border of the ventral horn. Still lower the sections show a small area to the right of the central canal, and slightly posterior to it, which is occupied by a fairly loose mass of lymphoid cells, similar to the infiltration described. Posterior to this and posterior to the central canal, extending into the gray matter of both dorsal horns, but more especially upon the right side, is an irregular area of hemorrhage, with small hemorrhages radiating from it and surrounding it. A little below this, in the upper dorsal region of the cord, the sections show nearly the whole central part of the cord destroyed by a diffuse and large hemorrhage, which is rather greater upon the right side of the cord than upon the left side, and leaves but little of the normal structures of the cord. The periphery appears normal, and near the borders of the hemorrhage there are occasional nerve cells, most of which appear fairly normal. Sections from the upper dorsal region, a little lower than this point, show a hemorrhage into the right dorsal horn, completely destroying it up to its root, but not extending into the white substance of the cord. There is great dilatation of blood vessels of the ventral cornua also, but no hemorrhages. Sections from this level, stained by Weigert's method for myelin sheaths, show a moderate diffuse degeneration of nerve sheaths, at the periphery of the lateral and anterior tracts, and of the lateral pyramidal tracts, slightly more marked on the right side than on the left. In other respects the cord appears normal, and also the pia, and nerve roots. Sections from the lower dorsal region show only a moderate diffuse degeneration of nerve sheaths in the borders of the lateral and anterior tracts, and in the lateral pyramidal tracts, rather more marked on the right side. Nothing else abnormal is seen.

Sections from the lumbar region show only slight degeneration in the lateral pyramidal tracts to about the same extent on the two sides.

Anatomical diagnosis: Glioma of the cervical portion of the spinal cord; hemorrhage into the tumor; hemorrhage into the spinal cord; secondary degeneration of the pyramidal tracts.

To resume, a boy of six years, after a trifling injury, developed two days later a weakness of the right hand and arm, with pain in the shoulder and with rotation of the head to the left, where it was held by muscular spasm. At this time there was weakness of the arm with retained electrical reactions, and no sensory changes, at least no marked changes. At this time a diagnosis was made of injury of the nerve roots either from the results of the accident or from disease of the vertebral arches. Later there was marked paralysis of the arm, with slight quantitative electrical changes and paresis of the leg upon the same side, with increased tendon reflexes. Fifteen days after this condition was noted the child suddenly died with symptoms of interference with the respiration, about seven weeks only after the first symptoms had been noticed, and five weeks after he had first come under observation. The autopsy showed a glioma of the whole of the cervical enlargement of the cord, into which hemorrhage had taken place, together with hemorrhage into the cervical and upper dorsal cord.

The case is unusual in the sudden termination by hemorrhage into the tumor, which is much more common in cerebral tumors than in those of the cord, though the gliomata of the cord are usually rich in blood vessels, and often show a brownish or reddish discoloration from the presence of small hemorrhages. In Mader's<sup>26</sup> case, spoken of above, death was also sudden, and he ascribed it to an acute edema of the tumor, which was of the dura. Saenger<sup>27</sup> reports a case of glioma which involved the whole of the lumbar region of the cord from the lower dorsal region to the conus, in which

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<sup>26</sup> *loc. cit.*

<sup>27</sup> "Ein intramedulläres Rückenmarks-tumor, Gliom." Münch. med. Woch., 1898, XLV, 978.



there was considerable improvement from the symptoms, but the patient died suddenly. Nonne<sup>28</sup> at the same meeting reported a case of spindle cell intramedullary tumor which extended from the upper cervical cord to the tenth dorsal segment, with total destruction of the cord in the cervical region, where death ensued with bulbar symptoms of disturbance of respiration and heart action. In this case the symptom first noticed was a weakness of the legs, with slight pains and diminished sensation, while later the paralysis of the legs became flaccid, and weakness of the upper extremities came on, accompanied by atrophy and quantitative diminution of the electrical irritability of the paralyzed muscles, and at the last, difficulty in swallowing, paresis of the left abducens, and choked disk. In this case there was a cavity in the part of the tumor which was in the cervical region and the fatal termination may have followed an increase of pressure within this cavity. Fraenkel<sup>29</sup> in his first case reports a gliosarcoma of the cervical region, in which the symptoms began as in this case, with weakness in the arm, and where the duration was short, only one and a half months.

Schlesinger,<sup>30</sup> from 135 cases of tumor from the Vienna hospital, found 20 intramedullary ones, and from 125 intramedullary tumors, collected from the literature and his own cases, he again found 20 of these to be gliomata. He states that glioma of the cord is more common in the cervical or lumbar parts of the cord than in the thoracic portion, while the reverse is true of the extramedullary tumors, which more frequently affect the thoracic part of the cord.

Our case was one in which there can be no doubt that there was present in the cord a true tumor formation, and not the condition described by Schultze, Schlesinger, and especially by Hoffmann, to which the name of gliosis has been given, and this both because of the very nearly complete replacement of the normal structures of the cord in those places where the tumor was most developed and because of its lim-

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<sup>28</sup> Münch. med. Woch., 1898, XLV, 978.

<sup>29</sup> Fraenkel, A., "Zur Lehre von der Geschwülsten der Rückensmarkshäute." Deutsche med. Woch., 1898, XXIV, 442, 457 and 476.

<sup>30</sup> *loc. cit.*, p. 7.

itation to comparatively a small portion of the cord, as well as the character of the new growth itself.

There is also in this case an apparent connection between a slight trauma and the development of the symptoms, which led us astray in the diagnosis. This connection of trauma with tumor of the cord or its membranes, however, has been often noticed, and trauma has been adduced as a cause of the development of new growths. Schlesinger<sup>31</sup> states that a review of the cases in the literature would in some instances allow the inference of the causal relation of trauma to the neoplasm, but that in other cases symptoms had existed previously in greater or less degree and the injury seems only to have acted as an accelerating cause, and, moreover, that in most of these cases we have to do with syringomyelia without tumor formation; that is, cases in which variations of pressure in the cerebro-spinal fluid could act with most effect. Schlesinger, however, concludes that in nearly all the more important groups of neoplasm of the cord and its envelopes, trauma in at least a portion of the cases seems to form an important etiological factor, either in the development of the new growth or in the hastening of the growth of an already existing one. However, in looking over the examples he cites in support of this view, one is forced to the conclusion that this statement is more evidently true when we have to do rather with growths proceeding from the vertebræ, or with the infectious granulomata, where a slight injury may determine the point of origin of the new growth, than with cases of glioma. Certainly it seems to me that our case can hardly be interpreted as affording support to any such theory, in view of the rapid course from the first appearance of symptoms and the marked extent of the tumor development, which seems too great to have taken place within the seven weeks which elapsed between the first symptoms and the death of the patient. I should be much more inclined to ascribe the first onset of symptoms in this case to a small hemorrhage into the tumor, perhaps produced by the fall, similar to the large hemorrhage found at the autopsy, which was probably the immediate cause of death.

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<sup>31</sup>*loc. cit.*, p. 101.

In this case it was exceedingly difficult to judge of the pathological condition causing the symptoms, which were practically root symptoms only, in spite of the fact that the tumor was intramedullary. Information which might have been obtained from the presence of bilateral paralysis was unattainable on account of the congenital absence of the left forearm, while sensory changes were slight throughout the course of the disease, and were certainly absent when the patient first came under observation, and it was only later in the course of the disease that paresis of the right leg showed that the pyramidal tract was involved; so that practically all the differential points which Starr gives<sup>32</sup> for distinguishing an intramedullary growth were absent in this case. These points are: the presence of Brown-Séquard paralysis before paraplegia; early and marked atrophy and reaction of degeneration; early trophic symptoms and bedsores; and the development of analgesia prior to anesthesia. However, the unfortunate lack of any late careful examination of the sensation precludes us from affirming positively that none of these things were present. In particular, Brown-Séquard paralysis may have been present and so have been a precursor of the paraplegia which we may presume would have developed later. Therefore it would be unwarranted to interpret this case as militating against the value of these indications, inasmuch as the early termination of the case prevented the development of the symptoms which in all probability would have given us some more definite clue to the nature and seat of the process with which we had to do, than it was possible to get up to the time of the sudden, and, I might almost say, premature death of the patient.

Any remarks on the nature and origin of gliomata are foreign to the scope of this paper, but it is worth while to note the early age at which the case occurred, though one of Schlesinger's cases was in a child of five years, but this was the only instance among the cases he tabulated in which glioma of the cord occurred under ten years, while the greatest number of cases occurred between the ages of twenty and thirty, and a search through the more recent literature has not revealed reports of any further cases in young children.

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<sup>32</sup>Starr, M. A., *Am. Journ. Med. Sciences*, Phila., 1895, CIX, 613.

## RECONSTRUCTION OF SERIAL SECTIONS OF BRAIN.\*

BY ADOLF MEYER, M.D., WORCESTER, MASS.

Dr. Meyer demonstrated several models of reconstruction of serial sections of brains, in drawings on superimposed glass-plates. The drawings were made by the help of the Edinger-Leitz apparatus. Higgin's inks had been fairly satisfactory. It is easy to calculate the relative proportions of thickness of section, thickness of glass-slide and linear enlargement and to get the correct proportions of width and depth.

The advantages of this procedure are:

a. Its quickness. The drawing of the models shown took only from three to fifteen hours each.

b. The lucidity of the result, especially the possibility of seeing the most tortuous courses of bundles, as the bundle of Vicq-d'Azyr and the mammillo-tegmental fibers, etc.

c. The ease of manipulation as compared to that of wax-models, which, moreover, are not translucent, and cannot be taken apart in levels for guidance in the study of the actual sections.

### DISCUSSION.

Dr. C. K. Mills said that although the time would not allow of any discussion he would like to say a word about these preparations. He had looked at some of them before the meeting, and it seemed to him they were unusually good for the purpose of teaching the structures, or the parts of structures, and the organs which have to be represented in different planes.

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123 "ZUR KÜNSTLICHEN FIXATION DER GELENKE BEI TABES" (Artificial Fixation of the Joints in Tabes). Adler (Neurol. Centralbl., Feb. 1, 1900. No. 3, p. 102).

Adler believes that the muscle hypotonus commonly observed in tabetics is in large measure accountable for their embarrassed locomotion. With this in mind he has tried fixation of certain joints, particularly the knee, by means of bandage-like appliances made out of woolen-elastic materials, with sufficient benefit to his patients to make him urge a more general trial of this simple procedure.

J. W. COURTNEY.

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\* These models were shown at the meeting of the American Neurological Association, May, 1900.

## EXPERIMENTAL WORK ON BRAIN PRESSURE FOLLOWING INJURY.\*

Done under the direction of William N. Bullard, M.D.

The experiments were performed at the Physiological Laboratory of the Harvard Medical School through the kindness of Professor H. P. Bowditch. The method employed has been to screw into a trephine hole in the skull a cylinder, into which is slipped another cylinder closed at the end next the brain with a thin rubber membrane and connected at the other end with a tube. This inner cylinder and the tube are filled with water, and the tube can be raised and lowered so as to keep the rubber membrane level, thus holding the brain in normal position.

The uninjured brain of an etherized cat will bulge through an opening left after removal of dura and bone, unless pressure is applied to keep it in place. The pressure necessary to prevent bulging varied in a typical case from 8 cm. to 16.9 cm. of water, according to the degree of etherization. The more ether given, the greater is the pressure until the anesthetic is pushed too far, when the pressure begins to fall. In the case mentioned, the average pressure during five hours of observation was 13 cm. of water. Every factor increasing general blood pressure has an instant effect on brain pressure. A cry (raising thoracic pressure), any general muscular movement, stimulation of the sciatic nerve, the giving of ether (increasing heart action), all demand a stronger pressure to keep the brain level.

Pulsations of the brain are much more extensive after hemorrhage, that is, the brain surface passes through wider excursions with the heart beat and the respiratory changes. Also after hemorrhage the pressure necessary to keep the brain from bulging is less, ranging in one case of considerable bleeding from 1.3 cm. to 4.7 cm. of water, with an average pressure during two and a half hours of 3 cm. of water.

After heavy blows on the skull the pressure needed to prevent the brain from bulging is greatly increased. In one

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\*Prepared for the twenty-sixth annual meeting of the American Neurological Association, held in Washington, May, 1900.

typical case the limits were from 10.1 cm. to 47 cm. of water according to the etherizing, the average pressure during four hours being 27 cm. of water.

Experiments thus far fail to show that there is any increase of general arterial pressure to account for the greater brain pressure after injury. No observations have, as yet, been made to ascertain if the increase is to any degree dependent on increased cerebral *venous* pressure.

These observations have confirmed those of Kramer, Polis, and Horsley, that the respiratory center is the first to be affected after a blow on the head. Many times when respiration has ceased artificial respiration for a few minutes has been followed by normal breathing which has continued for hours.

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- 124 "LA TOLERANCE DES BROMURES CHEZ LES EPILEPTIQUES AGES" (Tolerance of Elderly Epileptics to Bromides). Féré (Revue de Méd., Jan. 10, 1900).

Féré in this article states that potassium bromide is an anesthetic and amyothenic, and lowers vascular and nervous tension, diminishing the epileptogenic activity of the cortex and excito-motor power of the bulbo-medullary tract. If the other bromides are given in immediate high doses the same symptoms are produced. Individual tolerance varies extremely. A daily dose of 45 to 60 grains is enough to begin with. Bromic intoxication may readily supervene in organic disease of the heart. When used steadily, the drug tends to accumulate particularly in the brain and liver, but is found to some extent in all tissues. While in theory aged epileptics ought to show diminished tolerance to bromides, the reverse appears to be the case in many instances. Elderly epileptics are not uncommon, and with them the disease may have dated from childhood or developed only in late life. Senile epilepsy proper is due to various causes, alcoholism, moral shock, cerebral diseases, especially to arteriosclerosis. When due to this latter cause we may expect to find insomnia, vertigo, pre-cordial anxiety, etc., with intolerance to bromides. These patients may derive no benefit from bromides, and this condition forms a distinct contra-indication. It often happens, however, that elderly epileptics respond to small doses of bromides, from which they derive much benefit, attacks having been suspended by 4 or 5 gms. within a very short time. Others resist the effect of the bromides, exhibiting a tolerance, and this is the class which we wish to consider. No distinction is made between inveterate and recent senile epilepsy. Féré gives histories of ten cases of senile epilepsy treated successfully by bromides. This author regards it as important to determine the tolerance of bromides in every patient by a slow, gradual increase of the drug; intestinal and cutaneous antiseptics greatly favor tolerance. Contrary to most authorities, Féré thinks that most adjuvants to the bromides provoke intolerance more readily than the latter.

CLARK.

## A FORM OF SUBACUTE PRESSURE-NEURITIS.\*

BY THEODORE H. KELLOGG, A.M., M.D.,

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**Definition:** In default of any distinct literature, and in accordance with clinical observation, this affection is here defined as a subacute pressure-neuritis of branches of the brachial plexus, and it consists more especially in sensory and motor disorders in the region of the ulnar distribution.

**Etiology:** The immediate cause is pressure of the nerves involved, though it is possible that autotoxic and diathetic states may act as predisposing causes. Neither extrinsic poisons nor alcoholic or cachectic conditions were causative in the first cases of this affection, seen long years ago in hospital psychiatric practice. Patients violently homicidal or suicidal were then often confined in canvas strait-jackets with long sleeves crossed in front and tied behind, and with thick seams in the axillary region, causing compression and a direct mechanical origin of the neuritic trouble under consideration.

More recently the writer has known the pressure of a tight coat sleeve to produce a similar group of symptoms, which he has termed in common parlance "the coat-sleeve arm." These cases are not infrequently called rheumatic or neuralgic, but they improve only when the mechanical cause has been discovered and removed, and they are instances of subacute pressure-neuritis.

Analogous, but more severe, affections are the brachial paralysis neonatorum, the musculo-spiral paralysis potatorum, and the milder forms of traumatic injuries of brachial nerves, including pressure in crutch-paralysis.

**Symptoms:** In keeping with the severity or mild continuance of the compression, the onset of the symptoms may be sudden or gradual, reaching full height in a day or at the end of weeks. At first there is numbness of the little and ring fingers, with tingling and formication. Later there is pares-

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\*Prepared for the twenty-sixth annual meeting of the American Neurological Association, held at Washington, D. C., May, 1900.

thesia of all parts of the ulnar distribution. The pain may be considerable or insignificant, and finally well marked anesthesia may declare itself, along with an angiospastic state of the fingers, which may assume something of the appearance of "digiti mortui."

There is sometimes tenderness over the course of the ulnar, but sensitive points at the wrist and elbow are not present as in ulnar neuralgia.

Motor as well as sensory functions become involved in the more severe cases, and there is then difficulty of abduction and adduction, and also of flexion of the fourth and fifth digits. In extreme cases the musculo-spiral and the median nerves may also be affected, but a total arm palsy has not come within the writer's observation.

Clinical course: The average duration of this affection is some weeks or even months, if the original exciting cause was prolonged; and relapses are readily produced by renewed exposure to the special form of mechanical violence to nervous tissues.

Prognosis: The outlook for complete recovery is good after removal of the prime cause, though months will elapse before a cure whenever there is much anesthesia or any muscular atrophy.

Treatment: The radical part of the treatment is fulfilled by the ablation of the mechanical cause; and then hot applications, especially hot air, massage, electricity, and systematic exercise of the parts affected are to be patiently applied in accordance with symptomatic indications. If toxic substances are suspected as complicating etiological factors eliminatives, of course, have a rôle to play, and in every case the restoration of the general physical condition is all-important.



A CASE OF MULTIPLE NEURITIS WITH ATROPHY,  
FIBRILLARY TWITCHINGS, CRAMPS AND  
EXAGGERATED REFLEXES: TWO  
YEARS' DURATION AND  
RECOVERY.\*

BY WILLIAM JAMES MORTON, M.D.,

Professor of Diseases of the Mind and Nervous System and Electro-Therapeutics  
in the N. Y. Post-Graduate Medical School and Hospital.

The clinical symptom-picture of a severe case of multiple neuritis is still of interest, especially when unusual symptoms appear which would raise the question of a differential diagnosis between a peripheral and a cord disease. The following case presents unusual symptoms of interest.

Patient: Charles Rolker, æt. 40, civil engineer, contracted African fever at Fort Salisbury, Mashonaland, on February 7, 1892. The first and subsequent chills affected the left half of the body, apparently at least, for while the left half "felt freezing and in a chill," the right side felt perfectly normal. Took quinine, whisky, and antipyrine. By the 19th felt much better of his chills, and began to pan out gold, working with bare arms in cold water, panning five minutes and resting five minutes, often breaking out into profuse perspiration and feeling very weak between times. On February 20 he felt a rheumatic-like pain in the left shoulder; it was a sharp, gnawing pain, and kept him awake that night. The pain increased daily, until by the 26th he had sharp, shooting, darting pains, and he now carried his arm in a sling and wrapped in cotton, and could only sleep from 12 o'clock to 3 A.M., when the gnawing and darting pains commenced.

March 20 he started "down country," walking the first two hundred miles to save the jarring of the arm. By this time the fingers would swell, the skin became shiny and tingling, and he experienced a drawn feeling in the back of the neck. He had to be dressed and undressed by a servant, and also fed at table. Slight movements caused excruciating pains, and shooting pains came on irrespective of movements. At end of April another attack of chills and fever occurred. He had up to this time taken morphia in large doses, quinine and whisky, and because he thought that his malady was muscular rheumatism, as high as one hundred grains of salicylate of sodium in one day.

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\*Written for the twenty-sixth annual meeting of the American Neurological Association, held in Washington, May, 1900.

On May 11 he embarked at Capetown for England, and thence home to New York. The acuteness of the attack in the left arm and shoulder had subsided somewhat, but he now noticed both on board ship and upon arrival in New York city that the calves of his legs would tire quickly and would feel strained upon very light exertion. Also in the meantime he had frequent recurrent attacks of chills and fever.

The patient came under my care June 18, 1892, and was immediately put upon a course of quinine and tincture of red bark.

Examination: The left arm by this time was fairly comfortable, but it felt numb, dead, and without strength. Dynamometer: Right hand, 60; left hand, 50. Temperature,  $98\frac{1}{10}^{\circ}$ . Pulse, 76. The nerves of this arm are sensitive and thrill to the touch, and pressure at some points gives much pain. Over biceps the arm measures  $1\frac{1}{2}$  inches less than the right. On the whole the arm is quite helpless, and at times he suffers much pain in it. He is now more concerned about his legs than about the arm. The legs feel weak, stiff, sore, numb, and he can scarcely walk about; he had even resorted to going on his hands and knees to get about his room. No pain had yet developed, however, in the legs. A slight tap evokes a very highly exaggerated knee-jerk in both legs, each jump being followed by a succession of quick and vibratory movements of the quadriceps extensor, dying out to nothing. No ankle-clonus evocable. Patient states that the legs give big jumps in the night while he is in bed. No urinary or sexual troubles; no girdle sensation, and no sensory symptoms in legs.

On June 27, curious to relate, the exaggerated knee-jerk and the accompanying vibratory succession of fading-out jerks were absent, and the knee-jerk was about normal, but by July 7 and gradually the exaggerated knee-jerk again became established. Now also both tonic and clonic spasms of the legs increased to the extent of waking him frequently each night; he now also had priapism and frequent sexual desire with quick loss of sexual erection, but no troubles of urination.

By July 17, while less stiff and having less cramp, sensory symptoms developed in the legs and feet, with pains coming off and on. He also complained of pain in the sacral region of the back, in the calf, thighs, in the back of the neck, and at the tuberosity of the ischium on the right side, and it is painful for him to stoop, sit, or rise.

The patient continued under treatment during the summer and up to September 14, making marked improvement in the arm and legs, but unfortunately had persisted in long walks unknown to his physician, on the advice of another physician

"to walk his leg troubles off, as all he needed was exercise." The patient deeply rued this advice.

September 14 a series of cramps began in the muscles of the legs, arms, neck, abdomen, and feet; sometimes in one place, sometimes in another, and by day as well as by night; also he was easily fatigued both mentally and physically. Aside from this he felt comparatively well, and suffered almost no pain. Now also developed a new symptom, namely, a rapid atrophy of certain muscles, preceded by fibrillary twitchings in these muscles. When stripped and examined, September 19, it was found that the left supraspinatus was partially wasted, but was not sensitive; the left infraspinatus was atrophied so that the examiner's fingers impacted firmly against the scapula as upon a board, while on the normal side the scapula could not be felt and left the outline of the rhomboideus strongly marked. No spasms in this muscle nor fibrillary movements. Trapezius, both sides normal. Deltoids, normal. Biceps, right normal, left wasted to the bone, so that this region presented a concavity in contour rather than a convexity.

November 19 the electrical examinations for the reaction of degeneration had been frequently made during all this time of the invasion of atrophy with fibrillary twitchings and tonic and clonic spasms, and invariably gave the same result, namely, a progressive weakening of the cathodic contraction as compared to the anodic contraction up to a point where the anodic contraction began to surpass and finally greatly exceeded the cathodic, together with the usual qualitative changes and loss of reaction to faradism. Hundreds of observations corroborated this point in every invaded muscular area.

Since October 22 the patient had also developed all the characteristic sensory symptoms of neuritis in the right arm, and suffered some revival of pain in the left.

January 31, 1893. The patient's condition is now one where in addition to a neuritis of his right arm without atrophy, and in addition to a complete atrophic condition of his two spinali on the left side and the biceps on the left side, he exhibits throughout his entire muscular system fibrillary twitchings, now in this group, now in that, now in the legs, now on the abdomen or upper trunk muscles, or again the arms. These twitchings are visible to the observer, and resemble those of progressive muscular atrophy, with the exception that when violent they suggest to the patient "the beating of a pulse" or a sharp, small blow of a small hammer. These twitchings occur even at the mouth, and pull it back and forth; or in the breast, when the mammæ rise and fall like a feeling of trembling. They last six or seven seconds at a time. The patient

describes his condition as one of "jumps and flutters," namely, isolated muscle clonic spasms and fibrillary movements. He also has much insomnia.

January 13, 1894. Priapism has ceased and sexual functions are not far from normal.

From January to June the patient improved and was so much better that I sent him to Arizona to live entirely in the open air.

His infraspinatus and supraspinatus muscles had entirely filled out to their normal volume, as had also his biceps muscle, which now measured with the arm in extension  $10\frac{1}{2}$  inches, in flexion  $11\frac{1}{4}$  inches. At times he has mild attacks of "flutters," which are relieved by a dose of Carlsbad salts.

Six months later the patient wrote me that he was entirely cured and had resumed his avocation.

The treatment throughout was anti-malarial so far as that treatment was required, and by static electricity, in the form of the friction spark to the skin, for an effect upon centers by way of the afferent nerves, and in the form of a long, clean percussive spark delivered to the affected nerves and muscles.

The patient was seen in consultation with, or referred to, Drs. C. L. Dana and A. M. Starr several times, and they concurred in the diagnosis, though Dr. Dana at one time favored a view of progressive muscular atrophy, afterward coming back finally to the diagnosis of multiple neuritis.

It is to be regretted that the pathological changes at the bottom of such an attack are not likely to be easily forthcoming, since probably few such cases die. There can be but little doubt that changes in this form of multiple neuritis will be found in the cord as well as in the peripheral nerves, or at least changes in the cord roots. The case surely shows that multiple neuritis by no means exhibits a symptom-picture in actual practice as classical as might be inferred from reference to books.

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- 125 "ÜBER DIE WIRKUNG DES DORMIOL EINES NEUEN SCHLAFMITTELS" (On Dormiol, a New Hypnotic). Peters (Münchener med. Woch., 1900, No. 14, S. 463).

The author has used this new hypnotic (a combination of chloral and amylenhydrate) during nine months in 45 cases, among them 20 cases of diseases of the nervous system, both functional and organic. In 84 per cent. of all his cases the effect was satisfactory, and no unpleasant after effects were noted. He specially recommends it in the sleeplessness of functional neuroses. The chief advantages of the drug are its cheapness and its lack of unpleasant taste or odor. It is an oily liquid, and is given either in capsules or in a watery solution. The dose is from 0.5 to 1.0 grn. ALLEN.

## Periscope.

### CLINICAL NEUROLOGY.

- 126 ÜBER EPILEPTISCHE ACQUIVALENTE (On Epileptic Equivalents).  
Schultze (Münchener med. Woch., 1900, No. 13, S. 416, and No. 14, S. 465).

The author advances the view that the epileptic equivalent is more common and of more varied character than is generally supposed; that it may occur even where undoubted epileptic attacks are absent; that a complete amnesia for the period covered by the equivalent does not always occur, and that its absence does not necessarily exclude the diagnosis of epilepsy. He relates the following interesting cases:

*Case 1.* A man of thirty-seven, arrested for stealing two watches, was found to have had a history of having been—since an attack of scarlatina at thirteen years of age—periodically seized with an irresistible desire to wander off. At such times he would suddenly disappear, and would for days wander purposelessly about, returning as suddenly as he went. During his military service he on two occasions deserted without apparent reason, the last time being away for five and a half months before he voluntarily returned. For this offense he was sentenced to a year's imprisonment, as the military surgeons declared him sane and responsible. The patient declared that for one or two hours before he started out he had severe headache, "saw stars," or was dizzy, and sometimes had a sensation of something rising from his stomach; then he had no further recollection, until coming to he found himself in some strange place. He had never had a convulsion. Examination showed nothing special except a rather painful scar over the parietal bone, said to have resulted from a fall in early life. During a six weeks' stay in the asylum he had two attacks of vertigo, and at certain periods was noticed to be dull and depressed.

*Case 2.* An eighteen-year-old boy with a genuine epilepsy had as an aura sometimes an auditory hallucination (a spoken sentence), again a peculiar disturbance of memory (confusion of places and dates), and again double vision. At times the attack following these auræ was extremely slight, and even imperceptible, the patient not seeming to lose consciousness for even a second. The double vision was made out to be due to a fleeting abducens paralysis.

*Case 3.* A girl of seventeen years, having true epileptic seizures, during which she was often confused and violent. On some occasions before the attacks would suddenly expose herself, eat feces, etc.; on others would suddenly begin to ply her attendants with metaphysical questions, as "Is there a God?" "Is there a heaven?" etc., after which would come the attacks with complete loss of consciousness. The violence of the attacks, however, varied very much, and at times they failed entirely, being only represented by the conditions mentioned above. The patient then retained recollection of her acts and was much distressed thereby, but declared she could not do otherwise.

*Case 4.* A female epileptic had often as an aura sudden involuntary

discharges of urine, and again this symptom would occur with sudden pallor of the face, without the least loss of consciousness.

*Case 5.* A man of strong hereditary predisposition to insanity, during a fit of depression, shot himself in both temples, but did not die. Epileptic fits supervened, and his mental condition made his commitment unavoidable. He had very severe seizures, and at times, in connection with them, complained of a peculiar sensation in the left side of the face. Examination disclosed complete anesthesia of the region supplied by the first and second branches of the trigeminus, with blanching of the skin and development of wheals over the affected area. The condition would last several hours and would as quickly pass away. At times it would occur without any convulsion or disturbance of consciousness. The author is inclined to regard this as a case of reflex epilepsy, due to wounding of the trigeminus. ALLEN.

127 DIE ETIOLOGIE DER EPILEPSY (Statistics as to Etiology of Epilepsy). Lange (Psych. Wochen, 1899, No. 35-36).

The author follows Binswanger in making three classes of causes of epilepsy, viz., preparatory, predisposing and exciting. Of 741 cases studied, the proportion of males to females was 5 to 4. In the first decennium 53.4 per cent. were found affected for the first time, equally divided between the two sexes; in the second decennium 35.5 per cent. (more females) and only 11.1 per cent. of them were attacked after the age of 20 years. In 386 of the cases a predisposition was in evidence (303 inherited, 83 acquired). Of 303 inherited predisposition, 204 were of neuropathic antecedents, 63 toxicopathic (alcohol, syphilis, etc.); insanity or nervous diseases were present in the ancestry of 59 per cent.; epilepsy in blood-relatives occurred in 61.66 per cent.; epilepsy in parents in 23.75 per cent. The epileptic descent was more often from the mother's side; the father usually transmitted to the son, the mother to the daughter. With regard to acquired predisposition, the following casual moments are cited: Revaccination, 2 cases; alcoholism, 14; trauma, 28; sunstroke, 1, etc. Of determining causes are cited: Dentition, 29; puberty, 15; trauma, 24; intoxication or infections, 30; psychical shock, etc., especially fright, 65; miscellaneous, 18. CLARK.

128 KLINISCHE ERWÄGUNGEN AUS DER BEOBSACHTUNG SENSIBLER JACKSON ANFÄLLE (Clinical Considerations Derived from Observations of Sensory Jacksonian Attacks). A. Fuchs (Jahrbücher für Psychiatrie und Neurologie, Vol. XIX, No. 1, p. 1).

Sensory symptoms in Jacksonian epilepsy have always been regarded as accompanying manifestations of the motor phenomena. A. Fuchs attempts in this article to systematize the knowledge on this subject by an examination of the cases quoted in literature and by observations on eleven cases of his own. In regard to the etiology of the paresthesias we must at present assume that they depend upon the general cause of the disease itself. The phenomena of sensory Jacksonian attacks alone or as accompanying symptoms of the motor attack, are found in the following conditions: First, in the prodromal stage of progressive paralysis. They can in such cases appear as the first somatic symptoms of the disease. In one hundred cases their appearance was noted thirty-seven times. In five cases the sensory Jacksonian symptoms alone were present. Second, in diseased conditions of the brain which are limited in extent, such as tumors, abscess, cysts, etc. Third, in encephalomalacia. In this division observations are

limited. Fourth, hemicrania symptomatica. Krafft-Ebing found twenty-one cases in literature and three of his own. The conclusion seems justified that the sensory primary element of the central organ is much more easily acted upon than the motor element by noxious substances, and that in the ganglion cells, which have to do with sensory functions, the summation of stimuli first reach their effect, and by means of mere contiguity the motor explosion takes place. Sensory Jacksonian attacks are always indications of an anatomical central lesion. This symptom obtains a definite symptomatic importance when it accompanies hemicrania tardiva.

SCHWAB.

- 129 "EPILEPTIFORME ANFÄLLE IN DER RECONVALESCENZ EINES UNTERLEEIBSTYPHUS" (Epileptiform Attack During Convalescence from Typhoid Fever). Mühlig (Münchener med. Woch., 1900, No. 7, S. 221).

The author reports the case of a previously healthy man of twenty-three years, who after an attack of typhoid of moderate severity, having been free from fever for twenty days and while upon light diet and apparently doing well, had suddenly, at 3 A. M., a severe epileptiform attack, with loss of consciousness and convulsions, beginning with twitching in the little and ring fingers of the left hand. This was followed during the ensuing day by three similar attacks, after which the patient made an uninterrupted recovery, complaining of nothing more than some tingling of the two fingers of the left hand in which the twitching began. The urine was normal, as was also the heart; the author could find nothing to account for the convulsions. During a period of observation extending over a year he had developed no more attacks.

ALLEN.

- 130 CONVULSIONS POST-TRAUMATIQUE—EPILEPSIE (Post-traumatic Convulsions—Essential Epilepsy—Craniectomy). Mirallié (Arch. de Neurol., Mar., 1900).

Mirallié reports the history of a man thirty-six years old, vigorous, and having no neuropathic family history, who was trephined unsuccessfully for epilepsy. The man had fallen seventy feet and sustained a fracture of the cranium. Four years after recovery from this injury he developed epilepsy without tangible cause. Before each convulsion he would have an hallucination of seeing his dead friend. Some of the crises had a somnambulistic character; he had both grand and petit mal attacks. There was anesthesia on the right side of the body; special senses also were dulled on the right side. His only seeing eye, the right, was hemiopic (inner side); both optic nerves were atrophic. One hysterogenous zone was found in the right axilla. Cicatrix of old fracture was visible and evidences of depression were palpable in line of the old fracture. Patient was first trephined along line of scar; parts beneath depressed bone were compressed, but otherwise normal. A second trephine opening was made over the motor cortical center of the right arm. Bone-disks were not replaced and the meninges were sutured to the skin. There was improvement for five months, there being no major attacks during that time, but at the end of that time the major attacks returned and rapidly became worse than before. Mirallié joins the ranks of those who no longer believe in craniectomy for epilepsy. The case is of special interest from the fact that there was every reason to expect a different result than the one that occurred.

CLARK.

- 131 ACCIDENTS DE L'EPILEPSIE ET LA CONTRACTURE MUSCULAIRE (Accidents of the Epileptic Paroxysm Connected with Muscular Contraction). A. Féré (Revue de Chirurgie, Jan. 10, 1900).

The author states that the tonic contraction of muscle which plays so great a part in epilepsy may be attended with very different results. Contractions of the depressors of the jaw may cause luxation; the author reports an epileptic girl who dislocates her jaw with each paroxysm; the dislocation her mother can reduce. The largest bones in the body may break during a paroxysm; spontaneous fracture from muscular action has occasionally been reported. Abdominal hernia may result from spasm, but this is rare. The author has seen one case, a right inguinal hernia, in which the inguinal canal was unusually large. A tendency to hernia is frequent in all forms of degeneracy, but in epileptics the proportion is not excessive. Féré found on one occasion that 6 per cent. of epileptics were ruptured; later statistics made the number 9 per cent. Féré now describes a rare accident, *hernia of a muscle*. The patient was forty-five years old, and had for a number of years been in the hospital as an epileptic. The disease began when he was thirteen years of age. Patient is an alcoholic and has a nocturnal attack after each excess. At first view patient appears to be an entirely normal man, but on close inspection shows the following stigmata; cranio-facial asymmetry of the left side; chromatic asymmetry of the iris and anisocoria; pre-auricular fibrocartilage of the left side; bifid uvula; hypospadias; webbed toes on both feet. After a nocturnal attack he complained of pain in the leg, where a swelling was to be seen. This tumor was about the size and shape of a large almond, and was seated at the front of the leg at the junction of the middle and lower third. This tumor was reduced by placing the patient on his back with the foot flexed on the sole. The tumor was composed of muscle which had prolapsed through an opening in the fascia. Curiously on the opposite leg there was a similar defect in the fascia, but no hernia. Féré regards these breaks in the fascia to be evidences of imperfect or degenerate development. After a day or more the patient wholly recovered. In seeking for the presence of these muscular bosses, Féré examined 204 insane, and found that 15 per cent. had elevations of this character; in about one-half the cases they were bi-lateral. Féré concludes that these openings in the fascia of the leg ought to be looked on as congenital defects.

CLARK.

- 132 "MYOTONIE, EINE KRANKHEIT DES STOFFWECHSELS" (Myotonia, a Disease of Metabolism). W. v. Bechterew (Neurol. Centralbl., Feb. 1, 1900, No. 3, p. 98).

v. Bechterew believes that myotonia is, in the majority of cases, a disease which begins in early life, and not the result of any congenital abnormality either of the nervous or muscular systems. In his opinion the peculiar disturbances of muscle functionability which characterize the morbid picture are the result of a peculiar disturbance of metabolism whereby toxic products are liberated in the organism, which poison muscle tissue. This hypothesis he bases on the fact that in cases of myotonia he finds in the urine an increase of the products of muscle destruction, in the form of sarcin compounds and creatinin.

In all the author's own cases mechanical irritation of the muscles produced canaliculate contraction, and fibrillary twitchings of the small muscle bundles. This was most apparent in the deltoid. By com-



pression between the fingers and percussion with the hammer more or less pain was provoked in all cases, although any marked idiomuscular contractility was lacking, and this pain v. Bechterew interprets as a sign of altered muscular nutrition. In final proof of his views he points to the family tendency to myotonia, which is frequently observed, and says that the time of outbreak of the disease (*i e.*, whether it shall be congenital or acquired) depends simply upon the time at which the factors which lead to metabolic disturbances reach their development.

J. W. COURTNEY.

- 133 FALL VON ASTHENISCHER BULBÄRPARALYSE IN FOLGE VON AUTOINTOXICATION (A Case of Asthenic Bulbarparalysis Caused by Auto-intoxication). J. A. Feinberg (Neurol. Centralbl., Feb. 1, 1900. No. 3, p. 103).

The case here reported is most interesting. The patient was an intelligent man of 44, the father of six healthy children, and without hereditary or family neuropathological predisposition. Through sedentary habits and mental anxiety, he had acquired an atony of the lower bowel, which manifested itself in obstinate constipation. In the early part of November, 1898, the coprostasis became very marked, and in addition the patient experienced dyspepsia and nausea. Cathartics brought only temporary relief. In a few days there was a return of the obstipation, and purgatives were of no avail. The patient suffered from colic, meteorism, and vomiting. After five days of this deplorable situation there appeared threatening nervous phenomena—severe pain in the occiput and in the upper dorsal vertebræ, faintness, general weakness, paresthesia in all four extremities, bilateral ptosis, inability to wrinkle the forehead, paresis of the muscles of the eyebulbs, embarrassed lip and tongue movement, dysarthria, incessant drooling, difficult mastication, and dysphagia. No fever.

In great anxiety the patient, who lived at some distance, undertook the journey to Feinberg's clinic, but in the meantime he began to have profuse evacuations of the bowels, and following these the nervous symptoms diminished so rapidly in intensity that F. saw him only in the recovering stage—about a month after the development of the dyspeptic phenomena. Feinberg found: Slight bilateral ptosis, impaired mobility of the eyebulbs, wrinkling of brow impossible on right, slight left, paresis of the right facial. Speech and deglutition were considerably improved, the reflexes normal, and pulse and respiration showed no marked alteration. The cranial and spinal pain had disappeared and no abnormalities were discoverable in the internal organs, including the kidneys.

The patient complained of great weakness and prostration, even in bed, although there was no discoverable diminution in the size of his muscles and no trophic disturbance, and nothing suggesting gross lesion of peripheral nerves, cord, or brain.

By the middle of December the ptosis had disappeared and the lip movements were normal. The patient could wrinkle his brow fully, but the lower facial on the right was still parietic, and there was still a complaint of great general weakness. This weakness persisted for nearly a month after the subsidence of all local symptoms.

In considering the pathological substratum of the above condition the author excludes polioencephalitis superior, from the absence of its etiological factors and such stormy characteristics as disturbance of consciousness, delirium, hallucinations, and dyspnea. Alcoholic poison and other exogenous toxic agents he is also able to rule out. Hemorrhage into the bulb he does not believe probable from the age

of the patient, the integrity of his circulatory organs, the absence of nephritis, and the rapidly favorable course of the disease. His decision in the case is: Asthenic bulbarparalysis, without organic lesion.

J. W. COURTNEY.

- 134 "ZUR CASUISTIK DER RÜCKENMARKSVERLETZUNG DURCH WIRBEL-FRACTUR NEBST BESCHREIBUNG EINES GEHVERBANDES FÜR PATIENTEN MIT LÄHMUNG BEIDER UNTERER EXTREMITÄTEN" (Spinal Cord Injuries from Fracture of the Vertebrae, with Description of an Apparatus for Paraplegic Patients). Lenznick. Münchener med. Woch., 1900, No. 12, S. 386).

A man of twenty-six years fell from a scaffolding 14 meters high, striking on his back on a beam 3 m. above the ground, and then coming down upon his knees. He did not lose consciousness, but became at once paraplegic, with loss of control of his bowels and bladder. When he came under the author's care, a month later, the paraplegia persisted, there was complete anesthesia of the lower extremities, and there were bed-sores over the sacrum, the left malleolus, left leg and right heel.

There was deformity in the lower dorsal and upper lumbar regions, the twelfth dorsal spine being very prominent. Laminectomy of the eleventh and twelfth arches was performed by Prof. v. Eiselberg, a fracture of the left lamina of the twelfth arch being found, the dura and cord being compressed at this point and adhesions having been set up. The body of the twelfth vertebra was found prominent, but could not be reached on account of thickening of the dura, severe hemorrhage following at attempt to separate it.

The dura, being split up for some distance, was found to be adherent to the arachnoid on both sides, most firmly on the left. The cord showed a transverse scar. The dura was stitched and the wound closed. Healing took place promptly. The patient recovered some control over the bladder and rectum, but the paraplegia remained the same. In order to enable him to get about the author devised an apparatus consisting of two flat iron bars, each about 5 cm. wide, and long enough to extend from the shoulder to the heel. These were bent to fit the patient's figure, diverging somewhat above and below, one passing down behind each leg and being fastened together by three cross pieces at the upper part. These cross pieces were bent so as to partially encircle the trunk, and the leg portions were provided with four similarly curved supports, passing half way around the leg. To the lower ends of the bars were attached foot-plates, and to the upper straps for fastening the apparatus over the shoulders. This apparatus, thoroughly padded, was applied to the patient's back while he lay stretched upon his belly, and bound on with flannel bandages. The patient was then raised to an upright position, and eventually learned, with the aid of a Volkmann's bench, to get about over the smooth floor with a fair degree of ease. A picture of the original apparatus is appended. The author has since improved it by making the leg portions and the cross pieces for the trunk extensible, by a telescoping arrangement, and also has provided for the application of a head swing at the upper end.

ALLEN.

#### PATHOLOGY.

- 135 ZUR FRAGE DER "RETROGRADEN DEGENERATION" (On the Question of Retrograde Degeneration). E. Raimann (Jahrbücher für Psychiatrie und Neurologie, Vol. XIX, No. 1, p. 36).

Retrograde degeneration is a term used to describe the degeneration which takes place in a divided nerve centrally as opposed to the

peripheral or Wallerian degeneration. Raimann attempts to study this question by an examination of the cranial nerves. For the purpose of studying the peripheral neurone the motor cranial nerves are very suitable, as these nerves consist physiologically of the same kind of fibers, and as it is possible to follow the nerve and its root to their cells of origin. In this paper retrograde degeneration is used in its narrower sense and does not include the ascending degeneration which is thought by some to extend beyond the nuclei of origin. Dogs were used for the experiment. The facial nerve was laid free, and the three branches dissected and followed to their common root. Around the root a ligature was tightly passed. The nerve was then divided centrally; peripherally the stump was resected in order to prevent the possibility of regeneration. The wound was then aseptically dressed and the animals killed and examined in fourteen to fifty-six days later. The following conclusions are noted as the result of these experiments: 1. The peripheral portion of a nerve separated from its trophic center presents a very different appearance from the portion still in connection with this center. While the former shows, without exception, in its entirety the picture of Wallerian degeneration, the latter, together with the cells of origin, show evidence of a gradual and very slowly advancing destructive process, which must be described as atrophy. 2. The central portion of the nerve, as well as the cells, can undergo a rapid degeneration. This happens when traumatic or infectious or toxic processes complicate the break in the paths of conduction, for the reason that an injured neurone is always in a condition of more unstable equilibrium. 3. The term retrograde degeneration as applied to this condition is not a good one, and should be avoided. The author suggests the term traumatic destruction or degenerative neuritis. 4. While we must distinguish, as far as the central portion of the nerve is concerned, either an atrophy or a traumatic destruction, or a degenerative neuritis; in the peripheral portion only one process is observed, namely, Wallerian degeneration. SCHWAB.

#### THERAPY.

136 GEHIRN-SUBSTANZ IN NERVENKRANKHEITEN (Use of Brain Substance in Nervous Disease). V. Babes (Klin. ther. Woch., June 17, 24, 1900).

Following lines adopted by Pasteur in the treatment of hydrophobia similar results are claimed by the author following the use of injections of brain substance obtained from normal animals; further than this, functional nervous disease, such as neurasthenia and epilepsy, have been frequently influenced. This in the author's mind tends to show that the various toxins to which the nervous system is especially susceptible, and to which the symptoms of epilepsy, etc., are in main due, in the presence of foreign brain matter in the blood will combine more readily with this than with other nerve cells of the affected animal and thus will forestall such chemical changes as probably lie at the bottom of most functional neuroses. The author is certain that suggestion may be ruled out as an active factor in his cures, since he has been equally successful with children; and adults, where the subconscious self is clouded, have yielded not less remarkable cures.

JELLIFFE.

137 LE TRAITEMENT DE L'EPILEPSIE AVEC LES BROMIDES (Treatment of Epilepsy with the Bromides and Withdrawal of Sodium Chloride). Toulouse (Revue de Psychiatrie, Jan., 1900).

The author refers to a paper read before the Academy of Sciences by himself and Richet late in 1889, in which the conclusion was reached

that the deprivation of salt ought to raise the therapeutic potency of the bromides. The rationale of this action is the fact that bromides may be used to replace chlorides in the organic cells of the body. This cutting out of the chlorides from the diet is known as "hypochloruration." The young have more need of sodium than the adults. According to Bunge the latter take far too much salt in diet. Many ingest as much as 20 or 30 gms. daily, while 1 or 2 gms. are enough to secure parity between the potash and soda of the diet. With regard to hypochloruration in general, experimenters find that it is pernicious to withdraw salt entirely. But a man who weighs 130 pounds will get 14 gms. of sodium chloride on a daily average from his food. The lowest amount he can be made to receive by modifying his diet is 2 gms. Toulouse's diet to reduce the salt to the minimum is as follows: Bread (no salt added) 550 gms., salt .027; meat 280 gms., salt .684; milk 125 gms., salt .133; one egg 35 gms., salt .171. Making a total of 1.015 gms.

By allowing fresh beans or peas, and fruit in moderation, the salt is brought up to 2 gms. The amount of salt added in cooking, seasoning, etc., would bring this amount up to 8 or 12 gms. A similar diet arranged in four meals would be as follows: 7 A. M., milk, .25 liter (25 centiliters); 11 A. M., two cakes made with eggs, farina, milk, and sugar; coffee; 3 P. M., porridge made with farina, sugar, boiling milk, etc.; 5.30 P. M., bouillon, unsalted; boiled beef, unsalted; potatoes; no wine. This diet was acceptable to the patients. Twenty epileptics were treated by this plan; all were of essential epilepsy, and all exhibited enfeeblement of mind. Cases were selected in which convulsions were of frequent occurrence. Bromide of sodium was given because less toxic, and the daily dose, at first of 4 gms., was reduced in most cases to 2 gms. In most cases the results appeared to be strikingly beneficial. Patient No. 18, for example, who usually had an attack every five days, went 184 days without a solitary attack, and in all the cases a diminution of attacks amounted to 92 per cent. Those of the patients who used no bromides at all while upon the diet showed no improvement, showing that the diet is merely an adjuvant to bromides. CLARK.

## Book Reviews.

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CARE AND TREATMENT OF EPILEPTICS. By William Pryor Letchworth, LL.D., ex-President of the New York State Board of Charities, ex-President of the Eleventh National Conference of Charities and Correction, Author of "The Insane in Foreign Countries," "Children of the State," "Relief and Reform," "Homes for Homeless Children," etc. Illustrated. G. P. Putnam's Sons: New York and London. The Knickerbocker Press. 1900.

This work shows the advance made in the public care and treatment of epileptics, especially by the colony method, from the earliest conception of the idea in Bielefeld, Germany, to the present day in all countries, but more especially in the United States.

The first chapter consists of an interesting résumé of the history of epilepsy, its characteristics, heredity, the ages at which it develops, the influence of climate and race, and the ratio of epileptics to the population. In all the United States only five States have as yet established State institutions solely for the care of epileptics. These States are Ohio, New York, Massachusetts, New Jersey, and Texas. A number of States receive epileptics into their institutions for the feeble-minded. The fact, however, is gradually becoming recognized that institutions for the feeble-minded are not suited for the best care and treatment of epileptics. The writer considers the relation of the epileptic to general society, and the advantages accruing both to the patient and to the State and society from special and separate provision for this class of sufferers. He discusses fully all the details in the founding of a colony, from the preliminary action in gathering statistics to present to the Legislature to the education and employment of the patients. Many suggestions as to the selection of a site for a colony, as to the formation of boards of management, and as to the improvement of the grounds and arrangement and construction of the various buildings are presented. The question of classification of patients, and that of providing for their amusement, and many other important points in the thorough equipment of a colony in order to obtain the best results are considered.

In the following chapters the provisions made for the exclusive care of epileptics by each of the different States are presented. It is astonishing to note the immense amount of work that has been required in almost every State to bring about the passage of a law providing for the separate care and treatment of its epileptics. In Ohio, which was the first State to establish a State institution for the sole care of epileptics, the movement began in 1868, but it was not until 1893 that it was crowned with success and the Ohio Hospital for Epileptics opened for the reception of patients. This hospital, or colony, has a capacity of 900 patients, and there is a list of over 1,000 epileptics waiting for admission. Its grounds, buildings, and the work and provisions for the amusement and well-being of the patients are interestingly described, and some interesting statistics given.

New York was the second State to have a State institution solely for the care of epileptics, and Chapter III is devoted to its colony.

The Craig Colony for Epileptics, as the result of many years of untiring efforts, was opened for the admission of patients at the beginning of 1896, and has at present accommodations for 360 patients, but when buildings now being erected are completed the capacity will be increased to 720. There is already a large waiting list. The Craig Colony for Epileptics consists of 1,895 acres of land, which has been laid out under an extensive scheme of landscape gardening by experts. Great care has been taken that the colony should not have the appearance of an institution, but should be simply a village of small houses, each occupied by from ten, or twelve, to twenty-five epileptics. The present buildings and improvements in the grounds, as well as those planned, are interestingly described by the writer. A number of fine illustrations of Craig Colony accompany its descriptions.

In Chapter IV the provisions made by the States of Massachusetts, New Jersey, and Texas are described. The Massachusetts Hospital for Epileptics was opened for the reception of patients in 1898, and had 200 inmates within five months. There is a great demand for admission, far beyond the capacity of the hospital. Its grounds consist of 237 acres, of which one-half is tillable.

New Jersey plans a village, or colony, for the care of its epileptics, and in 1898 purchased 187 acres of land for the "New Jersey State Village for Epileptics." In 1899 the State made further appropriations for the purchase of more land and for the erection of cottages.

Texas also has recently secured 640 acres of land for the establishment of a colony for the care of its epileptics, and plans are being formulated for its development.

As already stated, the above five States are the only ones in which there are State institutions for the exclusive care of epileptics.

In Chapters V, VI, and VII the provision made for epileptics in twelve other States and in Canada are described. In these States the institutions where epileptics are received are state institutions for the feeble-minded, or for the insane, or, if for epileptics only, are supported entirely or mainly by private philanthropic associations. In all these States, however, the opinion is gaining ground that special and separate provision should be made for epileptics.

Chapter VIII treats of the provision made for epileptics in England, and the Home for Epileptics at Maghull, the colony at Chalfont, Saint Peter, the Meath Home for Comfort, and St. Luke's Home are described. These institutions are the results of private philanthropy. There is as yet no public provision for sane epileptics in England except in the poor-law work-houses and infirmaries.

It was in continental Europe that the first movement toward the organized care of epileptics originated, and in Chapter IX of this book Mr. Letchworth tells of the origin and progress of the movement in Germany, France, Switzerland, and other countries. He describes in full the Asylum for Epileptics at Biesdorf, the asylum at Zurich, and the famous Bethel Colony near Bielefeld. The Bethel Colony began its work in 1867, and has received since its opening 5,028 patients; it contains at present over 1,500 patients.

This book has appeared at a very opportune time, when all the countries of the world are beginning to give their attention to the movement in favor of the public and separate care of epileptics. The writer has shown a thorough and comprehensive knowledge of the subject, and has presented it in an interesting manner. The book will prove of great value to those countries and States which are planning the separate and public care of this class of sufferers.

The volume is profusely and finely illustrated, and as a product of the bookmaker's skill is to be commended.

BONAR.

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**Original Articles.**

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A CASE OF SO-CALLED LANDRY'S PARALYSIS, WITH  
AUTOPSY.<sup>1</sup>

By SIDNEY I. SCHWAB, M.D., ST. LOUIS.

Under the name of "Paralysie ascendante aigue" (Acute Ascending Paralysis) Landry in 1859 described a group of symptoms which has ever since been known by his name. Clinically the disease is characterized by a prodromal stage more or less marked, during which paresthesia, headache, malaise, etc., can be noted. Following this there develops an acute flaccid paralysis, involving first, as a rule, the lower extremities, then the upper and trunk muscles, and lastly the muscles supplied from the medulla. Sensation and sphincteric control are usually retained. The paralyzed muscles, although flaccid, show no evidence of atrophy and retain the normal electrical reaction. There is, as a rule, fever, but the sensorium remains clear unless the fever is excessive. The patient usually dies from paralysis of the respiratory muscles under the picture of suffocation. Landry himself could find no anatomical basis for this group of symptoms, and for a long time this negative finding was considered to be characteristic of the disease. In recent years, however, more careful study has shown a variety of pathological conditions. The variation in the clinical picture, to some extent, and the very dissimilar post-mortem findings, have led to considerable confusion in regard to this disease, so that to-day there is a well-marked tendency to do away with the

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<sup>1</sup>Read before the St. Louis Medical Science Club, June 12, 1900.

term Landry's paralysis, as indicative of a definite morbid type with a constant etiology, pathology, and symptomatology; and to use the term, if at all, as descriptive of a symptom-complex, the cause of which is at present unknown, the nature of which is problematical, and the pathology of which varies with almost every investigator.

In spite of this, however, as Strümpell says, the clinical picture of this disease is such a remarkable one that it is worthy of a generalized description, and it appears in all text-books as such. A very excellent summary of the present conception of the disease is given in Mills and Spiller's<sup>2</sup> article, an abstract of which, I think, will make this point clear. They say, first: The usual picture of Landry's paralysis is that of an ascending flaccid paralysis with little sensory disturbance, normal electrical reaction and retention of sphincteric control; its course is rapid and generally fatal. Second, there may be forms which depart from this, which resemble polyneuritis and myelitis to such a degree as to make the diagnosis difficult. Third, in some cases there may be no post-mortem findings, but these cases have probably been insufficiently examined. It may be also that in some cases death takes place so quickly that changes in the nervous system have not had time enough to develop. Fourth, Landry's paralysis may be classed as a myelitis. Fifth, a polyneuritis may exist. Anterior horn cell changes are found. It is difficult to determine whether they are primary or secondary in nature. Sixth, in some cases it is probable that the whole peripheral neurone is affected by the toxic process.

I shall not attempt in this paper to discuss the clinical aspects of the case from the standpoints of diagnosis, differential diagnosis, or treatment. This has been done often before, and very thoroughly. In Oppenheim's and Strümpell's textbooks will be found a very excellent clinical description as well as a summary of the post-mortem findings.

I wish, however, to call attention to a few considerations in regard to the etiology, which are well illustrated in the

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<sup>2</sup>"On Landry's Paralysis with Report of a Case." JOURNAL OF NERVOUS AND MENTAL DISEASE, June, 1898.



case which forms the subject of this paper. From the very beginning, Landry himself believed that the process was toxic in nature, for the reasons that the spleen was found enlarged, albumin was found in the urine, lymphangitis existed, and hemorrhagic foci were found in the lungs and other organs. Cases were then reported where this clinical picture followed, or was coincident with diphtheria, influenza, septicemia, etc. There is little doubt at present that in many instances, if not in every one, the etiological factor that is most significant is a toxin, differing according to the agent in question. Whether the organism penetrates into the spinal cord and medulla, and the toxin originated there produces its effect, or whether there is a generalized toxemia, affecting, for some reason not yet clear, the central nervous system, is as yet undetermined. One other point I wish to emphasize, and that is that there are some cases of peripheral neuritis, or rather multiple neuritis which run a fatal course, and have been confounded with the Landry symptoms. These cases should be classed as a neuritis and not as an ascending paralysis at all, for of course in a neuritis there are usually electrical changes in the muscles and nerves, whereas in Landry's paralysis none are present.

I am indebted for the material in this case, as also for the very complete clinical history, to Dr. F. Taussig, of the St. Louis Female Hospital, to whom I wish to express my thanks and appreciation.

Julia S., age 21 years, married, was admitted to the Female Hospital on November 16, 1899, with the provisional diagnosis of anemia. She gave the following history of her trouble: Born of healthy parentage, without any hereditary taint of any kind, she had not, previous to the present time, any serious illness except an attack of pneumonia. There was no specific history. Of the three children, however, to whom the patient had given birth, one was still-born, and two died immediately after delivery. The patient had not menstruated for seven months before entering the hospital, and had lost considerable weight. She said that about six weeks ago she noticed a tingling and sense of numbness in the fingers of the left hand; she did not, however, attach any importance to this. About one week later the same sensations were experienced in the fingers of the

right hand, and then spread up both forearms to the elbows. There was at this time no symptom of paralysis. Shortly afterwards the patient, by degrees, found herself unable to use her lower extremities, and for four weeks was confined to bed. Two weeks before entering the hospital, the patient was also partially unable to use her hands and forearms; there was no history of any general constitutional symptoms. When she was examined she appeared rather anemic but only slightly emaciated. She seemed rather dull mentally, answering questions slowly, but without any impediment of speech. Her disposition was however quite cheerful, in spite of her paralysis, and a tendency to insomnia was present. She ate heartily and was never nauseated. Her bowels were usually costive, but of late she had not been able to control fecal movements, so that when her bowels were loose, she had an involuntary passage. The same was true of her urination. Her incontinence was accompanied by burning on micturition, but no other abnormality. Cough had persisted for some time, but was not at all severe; expectoration was scanty. Her pupils reacted to accommodation and light; tongue was flabby and coated white, but without tremor or deviation. Chest, fairly well developed; lungs apparently normal on auscultation and percussion. Apex beat in fifth interspace; sounds clear and distinct. Lungs, spleen, liver, normal. Hyperesthesia of the abdominal wall was very marked, especially in the lower half. Vaginal examination was not made. Extremities: Some inability to move upper extremities, more marked on left side. Grip of both hands very weak, especially left one. Lower extremities absolutely flaccid and paralyzed. Knee-jerk absent on both sides. No sensory disturbance of pain, tactile sense or muscular sense in any of the extremities. No trophic changes in skin or marked atrophy of muscles. Urine analysis: Specific gravity 1023, acid reaction, light amber color, no albumin, no sugar. Treatment: Strychnine, iron and iodide of potash.

During the first nine days that the patient was in the hospital, no marked change was observed. The incontinence of urine and feces persisted, and she complained of some pain in the upper extremities. Her appetite remained good. On the tenth day she said she was feeling worse, saying that she had a spell during the night when she could hardly catch her breath. This attack was rather severe but of short duration. No more attacks appeared during that day, but she complained of feeling weak. The following morning at six o'clock she told the nurse that she was feeling better, and ate

a hearty breakfast. At eight o'clock she was suddenly seized with severe dyspnea. She was found to be cyanotic and gasping for breath, but with a comparatively strong, regular, and infrequent pulse. Atropine 1-120th of a grain was given hypodermically but at 8.40 o'clock she died.

Autopsy: Considerable emaciation; rigor mortis not set in. Left pupil slightly larger than the right. Scaling epidermis about the calves. No edema. Chest: Pleuritic adhesions on right side and laterally, also posteriorly on right side to diaphragm; no adhesions on left side. Right lung showed two nodules, caseous in nature, in the apex, about the size of a large pea; similar nodule in left apex. Lungs otherwise normal. Heart showed liquid blood in cavities, no blood clots. Heart muscle cuts soft and is friable; some fatty degeneration; valves all normal. Aorta shows spots of hyaline degeneration. Abdomen: Very little adipose tissue in abdominal wall and omentum. Spleen slightly enlarged; capsule not tense, but substance very soft. Kidneys show congestion; pyramids prominent; cortical markings pronounced; capsules not adherent. Liver congested with dark blood, but not otherwise abnormal. Intestines show no signs of inflammation externally, but lymph follicles in ileum somewhat enlarged and reddened. Two round worms found in the intestine. Uterus retroflexed, ligaments very lax, no signs of inflammation. Ovaries and tubes normal.

This very complete history is typical enough to class the disease under the symptom-complex described by Landry. The diagnosis need not detain us, as the important point at issue is not the name of the disease, but what condition can produce such a group of symptoms as is reported in this history. To be remembered in the post-mortem findings are the condition of the spleen, the old tubercular lesions in the lungs, the pleurisy, and the acute hyperemic kidney. These are directly in line with the assumption of the toxic etiology of the disease, which Oppenheim insists upon very strongly.

The brain and cord, macroscopically, showed nothing abnormal. Upon section of the cord evidences of marked hyperemia could be seen; so intense was this in fact, that it was thought at first that there might be here an acute myelitic process. Upon closer examination it was discovered that this condition depended, as far as could be determined by the naked eye, upon a tremendous capillary engorgement

of the whole cord and meninges. Microscopic examination showed the correctness of this assumption.

The macroscopic section of the brain showed two areas of softening, bilateral in distribution, involving the internal capsule at about the level of the lenticular nucleus. This was regarded as due to an injury to the brain in removing it from the skull. No evidence of meningitis was found, the membranes being somewhat thickened, but everywhere free. No other gross lesions could be demonstrated. The cord and portions of the brain, medulla and cerebellum were placed in 10 per cent. formol and in Müller's fluid. The sections of the cord and medulla were placed alternately in Müller's fluid and formol, so that Nissl's stain could be employed on sections approximately from the same level. A portion of the left sciatic nerve was also placed in Müller's fluid, for examination for possible neuritis. The result of these examinations will be given later.

In examining the literature of Landry's paralysis, I have noted only those cases in which a post-mortem examination is given. I shall not attempt in this paper to give a complete bibliography, but merely to indicate the post-mortem findings in the cases recorded in the last three years and so compare them with the findings in this case.

Boinet,<sup>3</sup> in a typical case of Landry's paralysis, with death, similar to this one, found vacuolization of the nerve cells in the cord and thickened spinal meninges. Sciatic and median nerve showed evidences of a degenerative neuritis. A few scattered bacilli were found in the cord.

Mills and Spiller<sup>4</sup> found evidences of polyneuritis. In many ganglion cells of the anterior horns throughout the whole spinal cord, Nissl stain showed central chromatolysis and migration of the nucleus. The question in this case was whether the pathological process in the cells or in the peripheral nerves was to be regarded as the primary source of the condition.

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<sup>3</sup>"Un cas de paralysie de Landry." *Gazette des Hôpitaux*, 1899. *Ref. Neurologisches Centralblatt*, Feb. 15, 1900. No. 4.

<sup>4</sup>*JOURNAL OF NERVOUS AND MENTAL DISEASE*, 1898, No. 6.

Goebel,<sup>5</sup> in a paper read before the Aertzliche Verein in Hamburg, in January, 1898, entitled "Zur pathologische Anatomie der Landry'sche Paralyse," brings out very clearly the point that was before indicated in regard to neuritis, especially the neuritis acutissima progressiva of Eichorst. He says that our conception of Landry's paralysis should be limited to those cases which conform to the original description of Landry himself. In this case the following pathological changes were noted: Peripheral nerves normal. In the cauda equina some bundles of nerves in the neighborhood of hyperemic blood vessels were found degenerated. There was an increase of interstitial substance without an increase of nuclei. By Marchi's method, slight degeneration was found from the decussation in the medulla to the oculomotor nucleus. The spinal cord was mostly intact. Bacterial examination of the spinal cord and sections stained for organisms were negative. Nissl's stain showed no certain changes.

Giraudeau et Leopold Lévi.<sup>6</sup> Careful examination of the spinal cord with Nissl's stain showed absolute normal cells. Peripheral nerves and anterior roots were also normal. Medulla cells also intact.

F. von Reucz.<sup>7</sup> Hyperemia of the whole central nervous system. Nissl's stain: degeneration of the motor cells of the anterior horns and cells of the motor nuclei of the medulla. Slight degeneration of the spinal cord fibers. The vessels of the pia showed round-cell infiltration and hypertrophy of the intima. No bacteria. The vessels seemed similar to those found in endarteritis syphilitica.

L. B. Wilson and J. L. Rothrock.<sup>8</sup> Bacteriological examination negative. Slight congestion of the blood vessels of the cerebellum and cerebrum. Walls of the large vessels of the medulla infiltrated with leucocytes. Many nerve cells were found swollen, with absence of granular structure and migration of nucleus. No degeneration of the fibers in the

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<sup>5</sup>Ref. Neurologisches Centralblatt, April 1, 1898.

<sup>6</sup>Revue Neurologique, p. 669, 1898.

<sup>7</sup>Charité Annalen, Vol. 23, p. 317.

<sup>8</sup>Phil. Medical Journal, p. 1181, 1898.

medulla or cord. Median, intercostal, and sciatic nerves degenerated. The process was to be regarded as a parenchymatous ascending polionmyelitis, which originated centrally and extra-vascularly. The peripheral nerve changes were to be regarded as secondary.

W. L. Worcester<sup>9</sup> found in the large cortical cells migration of the nucleus and swelling. The medulla was normal. A few cells in the lumbar region were pathologically changed; the majority of cells normal. Slight varicose swelling of the peripheral nerves.

J. W. Thomas.<sup>10</sup> Acute inflammatory exudate of the anterior horns; parenchymatous degeneration of the ganglion cells with their dendrites; infiltration of the perivascular spaces of the anterior horns; slight infiltration of the posterior horns around the blood vessels and in the white matter; slight degeneration of the fibers. Anterior and posterior nerve roots were degenerated. Micro-organisms were not demonstrated either in sections or by culture. Second case: Degeneration of all peripheral nerves examined; motor cells of the anterior horns stained by Nissl show degeneration; other cells intact; no micro-organisms.

Mills and Spiller.<sup>11</sup> Marked hyperemia of the whole central nervous system; myeline degeneration of the peripheral nerves; nerve cells of the anterior horn swollen, with migration of the nucleus and chromatolysis; Clarke cells normal; no micro-organisms.

Piccinno F.<sup>12</sup> Vesicular degeneration involving the nucleus and cell protoplasm; bacteria found, not only in the vessels and perivascular spaces, but also in the protoplasm of the nerve cells. These organisms were oval cocci, found mostly in groups, and sometimes in short chains.

Hertz and Lesne.<sup>13</sup> Thickening and infiltration of vessel walls; peripheral nerves intact; ganglion cell changes dependent upon the condition of the vessels.

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<sup>9</sup>JOURNAL OF NERVOUS AND MENTAL DISEASE, Vol. XXV, No. 5.

<sup>10</sup>Am. Journal of Medical Science, viii, 1898.

<sup>11</sup>*Loc. cit.*

<sup>12</sup>"Su di un caso de Paralisi Landry." Ann. d Neurologia, vol. 15, No. 1, 1897.

<sup>13</sup>"Paralysie ascendante aigüe experimentale." Société biolog., 23, No. x.

Remlinger.<sup>14</sup> A coccus isolated from a case of septicemia; pure culture obtained and injected into rabbits. Acute paralysis developed, followed by death in five days, from paralysis of respiration. Cultures from the cord gave the original cocci; hyperemia of the cord, especially in the lumbar region and the gray substance.

Courmont et Bonne.<sup>15</sup> Peripheral nerves normal; vacuolization of the anterior cells; hyaline degeneration; nucleus of the hypoglossus affected, bacteriological examination of the cord and cerebro-spinal fluid showed the presence of a diplococcus, similar to the streptococcus.

Knapp and Thomas.<sup>16</sup> Marked dilatation of the blood vessels, but without hemorrhagic extravasation; degeneration of the nerve roots by Marchi's method; diffuse degeneration of the white substance of the cord; anterior cells by Nissl show few normal ones, and in some of them no nucleus or nucleolus is found; posterior cells show few changes; fatty degeneration of the sciatic nerve; bacteriological examination negative.

I shall not attempt to critically analyze in any detail these findings. Knapp and Thomas in the article referred to have done this very successfully. On account of the multiplicity of the microscopic reports the task of presenting a clear résumé of them is very difficult. In a general way they may be summed up as follows:

In a few cases organisms have been demonstrated in the cord and have been cultivated from it, or from the peripheral nerves or spinal fluid. These organisms show no constancy, and from an etiological standpoint they can have at present no definite significance. In a considerable number of cases the peripheral nerves have shown evidence of a neuritis. The general opinion seems to be that this neuritis is secondary to the process in the cord, and bears no definite similarity to a polyneuritis, or not enough, at any rate, to

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<sup>14</sup>"Paralysie ascendante aigue." *Presse médicale*, 12, iv, p. 209.

<sup>15</sup>*Archives de Neurologie*, Nov., 1899, pp. 354-373.

<sup>16</sup>"Landry's Paralysis." *JOURNAL OF NERVOUS AND MENTAL DISEASE*. Feb., 1900, No. 2.

regard Landry's paralysis as a variety of neuritis. In a majority of all cases very definite and significant changes have been found in the nerve cells of the cord, mostly localized in the anterior horns. These changes point, without doubt, to a pathological process in these cells. The cells of the posterior horn have, as a rule, been little affected. The changes in the nerve cell are the changes that we see from acute inflammatory processes affecting the central nervous system, such as are found in acute myelitis, epidemic meningitis, etc. The most constant finding is, perhaps the hyperemic condition of the cord and membranes.

Evidences of degeneration of the white matter of the cord are not constant enough to be important. A few cases have shown no abnormal changes at all, and in a few cases, together with abnormal changes in the cord elsewhere, the nerve cells have been found normal. The absence of acute inflammatory processes, in the way of cell infiltration or softening, is very significant. The most definite statement in regard to the pathogenesis and pathology of the disease is found in Knapp and Thomas's<sup>17</sup> article, which is that the process is an acute parenchymatous degeneration involving the peripheral motor neurones, arising from some toxic or infectious cause. Mills and Spiller came independently to the same conclusion.

This is certainly very strong evidence, and it is with considerable hesitation that I am forced, as a result of a study of my specimens, to adopt another view, and to regard the process, in this case at least, as primarily an interstitial one, which finds its expression principally in the abnormal condition of the blood vessels.

Sections of the cord, medulla, cerebellum, and of various portions of the brain cortex were stained by the methods of Nissl, Weigert, Pal, Van Gieson and with picro-carmin, hemalaun, etc. The sciatic nerve by Van Gieson, carmin and Weigert. It is to be regretted that it was impossible to make use of Marchi's method. The result of my findings may be tabulated as follows:

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<sup>17</sup> *Loc. cit.*



Nerve Cells; Anterior horns; Nissl staining: For the most part normal in staining qualities and in shape. Nissl bodies show the usual character. Nucleus and nucleolus normal. Cell outline regular and dendrites can be followed the usual distance. The number and size of the cells appear normal. Some cells show pigment. Here and there possibly one cell in five shows slight chromatolysis, mostly central in variety. Vacuolization of the nucleolus occasionally observed. No irregularities of nuclear membrane. In a very few scattered instances migration of nucleus and total disappearance of the same were found. It is to be noted that the Nissl stain was made as freshly as possible and studied at once. After a lapse of almost two months, they were again studied, and it was noted that the pigment was much increased.

Cells of the posterior horns including Clarke's cells: Nissl staining. Most of the cells show the usual normal picture; in a few instances the usual accidental chromatolysis or slight variations from the normal was observed.

Medulla, cortex, cerebellum. Nissl staining: Cells show no definite pathological changes, being very similar to the condition found in the cord. The cells of the nuclei of the cranial nerves seem normal. It was observed, however, that the cells of the hypoglossus nucleus seemed to show more variation from the normal than the others. Nerve cells from all these regions were also studied in carmine and in Van Gieson preparations. The impression of their normal condition, as demonstrated by the Nissl stain, was strengthened.

Peripheral nerve: The only peripheral nerve that was saved for examination was a portion of the left sciatic. This was found to be normal in every particular.

Anterior and posterior nerve roots: No pathological changes were found in the nerve fibers.

Meninges: They were found everywhere very hyperemic and, in places, thickened, though not adherent. The blood vessels from the pia as they entered the white substance of the cord were much more tortuous and more numerous than normal. In many places the vessels were congested with blood, which was shown very clearly by the Van Gieson method.

Cord, as a whole: No evidence of degeneration in the white matter of the cord was found either by the Weigert stain or by its various modifications, all of which were used. One exception to this was found in a few sections in the upper dorsal region, where slight V-shaped degeneration, with the point of the V anteriorly, could be followed for a short distance. This was regarded as of little importance, being probably due to faulty sectioning. No evidence of degeneration was found in the white matter of the medulla, pons, *cruræ*, cortex, or cerebellum. I wish to mention here that, as the Marchi method was not used, this point cannot be regarded as definitely settled in this case, as very possibly there may have been degenerated areas in the cord too delicate to be demonstrated by the methods employed.

Blood vessels: As it appears to me that the primary and most important changes in this case were found in the blood vessels, I shall attempt to describe their condition in some detail. The intense and generalized hyperemia of the whole cord was touched upon in the macroscopic description of the specimens in the beginning of the paper. The reason for this is apparent in the sections studied under the microscope. The blood vessels are thickened and tortuous, and their ramifications can be followed out to an extraordinary degree. At first sight their number seemed to be increased (*Gefässwucherung*), but it is very probable that, owing to the enormously increased injection of the vessels, more of the minute capillary branches are made visible by the stain than would otherwise be the case. The injection of the blood vessels is as marked in the posterior as in the anterior portion of the cord, and between the white and the gray matter no general difference could be observed. The vessels in the cerebellum, medulla, pons, and to a very slight extent the vessels of the cerebrum, showed the same condition of active hyperemia. It was very marked in the medulla and cerebellum. The vessels cut crosswise or longitudinally were found filled with blood, the same condition being found also in meningeal vessels and the vessels of the anterior and posterior nerve roots. The caliber of many of the vessels was enormously increased, so

that in the posterior nerve root, for example, a blood vessel occupied one-third of the space in a section of the root. In many places, apparently anomalous vessels were to be found, for instance in a number of sections from the dorsal cord the vessel running into the posterior commissure gave off a large-size branch about one-third of the way down anteriorly, the branch taking a diagonal direction towards the lateral column. Vessels of large caliber were seen in the neighborhood of the central canal surrounded by a clear space. Many of these vessels were of striking size.

**Hemorrhage:** Free blood was found everywhere in the cord for the most part in the form of small punctate hemorrhages, but sometimes of considerable size. The hemorrhagic foci were of greater size in the gray matter than in the white, and were more frequent in the upper regions of the cord than in the lower. The medulla, in the neighborhood of the hypoglossus and vagus nuclei, showed the presence of numerous hemorrhages. Most of the hemorrhages were of quite recent date, the form and arrangement of the red corpuscles being retained. Others were evidently older, and in a few places pigment granules from degenerated corpuscles were present.

**Vessel walls:** A careful examination of the vessel walls was made for the purpose of determining the presence of degeneration. Although some vessels, especially those in the neighborhood of the anterior or posterior fissures, showed slightly thickened walls, the majority showed the reverse condition; the walls being thinned by the tremendous pressure of the overfilled blood vessels. No evidence of degeneration of the vessel walls could be found. No increase of nuclei and no endothelial infiltration could be observed. The perivascular lymph spaces were found filled with blood, and slight evidence here of an endothelial proliferation was seen.

To sum up these findings: Nerve cells were found normal, or at any rate the slight variation found in them could be explained by preagonal or post-mortem chromatolysis. Absence of degeneration in cord. Absence of neuritis. Absence of myelitic process, of softening or purulent inflammation, and of meningitic process. The positive findings were limited to the

blood vessels and the perivascular lymph spaces alone; these consisted in a tremendous vascular congestion, a thinning of the vessel walls, due to this increased pressure; and hemorrhages with escape of free blood into the nervous structure, and with the possible increase in the number of vessels. All this with an absence of inflammatory product, and an absence of positively demonstrable diseased condition of the vessel walls other than the thinning before alluded to.

I wish to call attention to one possible source of error in regard to the hemorrhages, which is worthy of consideration. Is it possible that these hemorrhages are artefacts, produced by a mechanical destruction of the overfilled blood vessels in the process of sectioning? Could the blood, so to speak, have been pressed out of the blood vessels by the mechanical force of the microtome knife? This might be possible if it were not plainly demonstrable that there was often a break in the continuity of the vessel where the hemorrhage had taken place; further the evidence of slight compression of the nerve sheath in the neighborhood of the hemorrhages could not be explained in this way. Again, the hemorrhages are found too frequently and are quite independent of the plane in which the sections were cut, and there is evidence of the long duration of some of the hemorrhages.

In the face of these findings then, there seem to be two theories, both of which, it is true, only partially account for the symptoms in the case, and both of which must be regarded as unsatisfactory: first, a mechanical theory. The symptoms may be due to the effect of the pressure of the free blood upon the nervous structures which lay in their immediate neighborhood. In favor of this idea is the number of these hemorrhages, their distribution, and their greater prevalence in the upper portion of the cord. Against it are the greater number of hemorrhages of recent date, and an absence in a marked degree of the effects of old hemorrhages, as fibrin, blood crystals, pigmentation of surrounding structures, etc.

Second, the toxic process, whatever its nature, produced in the cord a condition very similar to that of acute non-purulent encephalitis. The nerve cells of the anterior horn were affected,

but to such a minute degree that their morphologic appearance could not be regarded as differing from the normal when studied by the Nissl stain. In other words, they are to be regarded as functionally affected. The absence of neuritis or degeneration in the anterior or posterior nerve roots would appear to strengthen this view. To explain the purely motor character of the symptoms, it is to be remembered that the anterior horns are more freely supplied with blood than the posterior, and that the purely posterior sensory cells are removed by their position from the direct effect of either a toxic or mechanical influence. The fever and constitutional symptoms are, without doubt, due to the original toxic process, which may have had its origin in the old tubercular foci in the lungs.

While not in any sense doubting either the possibility or the correctness of Knapp and Thomas' assumption of the parenchymatous nature of this process, yet in my case of Landry's paralysis the primary change must have been an interstitial one. I am strongly of the opinion expressed by Taylor and Clark,<sup>18</sup> in a recent article on the subject, that Landry's paralysis is not a definite clinical type, but that it is an acute ascending paralysis comprising a group of symptoms depending upon the most varied causes, and showing such variety of pathological appearances that the term has merely a clinically descriptive value, in much the same way that hemiplegia, paraplegia, or any other term used to describe a clinical picture may have.

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<sup>138</sup> UEBER EIN NEUES SCHLAFMITTEL AUS DER GRUPPE DER URETHANE (A New Hypnotic Belonging to the Urethane Group). Paul Schuster (Therapeutische Beilage der Deutschen med. Wochenschrift, June 7, 1900, p. 19).

Schuster finds hedonal very useful in cases of functional nervous disease with insomnia. He employs it in doses of two grams, and in the thirty-eight cases in which he has used it has seen no bad results. Sleep follows the administration of the drug within a quarter to a half hour, and lasts from five to seven hours. None of his patients had to urinate more frequently than they did before they had taken the drug.

SPILLER.

## AN ATYPICAL CASE OF MULTIPLE SCLEROSIS.\*

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Foundation.

Mr. D. was examined by us at the Home for Incurables, in September, 1899. We saw him once only. He stated that he was forty-six years old, single, and with no vicious habits. He denied having used alcohol even moderately. His health previous to his present illness had always been good and he had never had any venereal disease. The onset of his present trouble was slow; its course progressive and steadily downward. Seven years ago he began to have difficulty in walking, and now cannot move his legs at all. He has not been able to walk or even stand for several months. He has never had any pain in the legs. He has, at times, had slight spasmodic retention of urine, but has never been catheterized. During the past year his bowels have been moved only after the use of purgatives.

Examination.—He is a man of excellent build, chair-ridden, and unable to move either leg, as much apparently on account of spasm as because of palsy. The spasm in the legs is greatly increased by attempts at passive movement. The knee-jerks are very much exaggerated, ankle clonus is present, and the plantar and cremasteric reflexes are very marked. A tap anywhere on either leg causes a clonus to appear in the whole extremity. Motion in the arms is good (later he complained that the arms had become a little weak), and the grip is strong. There is no ataxia nor rigidity in the arms. Sensation is normal over the entire body. There are no cranial nerve palsies. The heart and lungs are normal. There is no wasting of the legs, indeed the legs are, if anything, more developed than the arms. The left great toe is in constant extension and the left foot is inverted on the ankle. There are no trophic changes of the skin, joints, or nails. There is no difficulty in speech or mental defect. Gastric or other crises, headache, girdle sense, vertigo and vomiting are all absent.

Dr. Charles K. Mills at one time treated the patient, and has kindly sent us the following notes: "The first notes were

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\*Read at the twenty-sixth annual meeting of the American Neurological Association, May, 1900.

made June 12, 1895. For three years previous to coming under observation he had been annoyed at times by numbness both in his left and right hands, but more marked in the former, and he had had increasing difficulty in doing such acts as buttoning his collar, especially with his left hand. He had also gradually lost the power of properly controlling the movements of his lower limbs, especially of his right leg, which, as he then expressed it, showed a tendency to flop around. He found it necessary to see the ground in order to walk without staggering. He could not balance himself well, even with his eyes open. He was weak in the legs and, as compared with his condition of a year or two previous, he was neurasthenic and easily exhausted. He complained that he could not hold his urine as well as he had done, and of some haziness of vision; he had no diplopia, however. No history of lancinating or other pains could be elicited, and he had no sensory symptoms except the numbness above described. Touch, pain and temperature sensations were preserved. Knee-jerks were exaggerated, and ankle-clonus was probably present, although I did not discover this until a subsequent visit. Briefly, the symptoms presented by the patient at the time of my first examination were: Moderate ataxia in both the lower and upper extremities, a general neurasthenic state, exaggerated, deep reflexes and absence of all sensory symptoms except some numbness in the hands, especially in the left. I saw the patient at intervals in 1896 and 1897, and again a few months before his death, in 1899. In March, 1896, I examined him, in consultation with Dr. S. Weir Mitchell. At that time he had the symptoms above described, with marked ankle-clonus. He grew more and more ataxic and paretic, and finally was compelled to give up active employment, probably in the latter part of 1897, although I am not sure as to the time. It is interesting to note that although he was markedly ataxic and paretic during 1896 and 1897, spasticity was not present, or at least was not of a decided character. When I saw him a few months before his death he was helpless, emaciated, with marked spasticity and contraction of his limbs, although in full possession of his intelligence."

He died suddenly a few weeks after our examination.

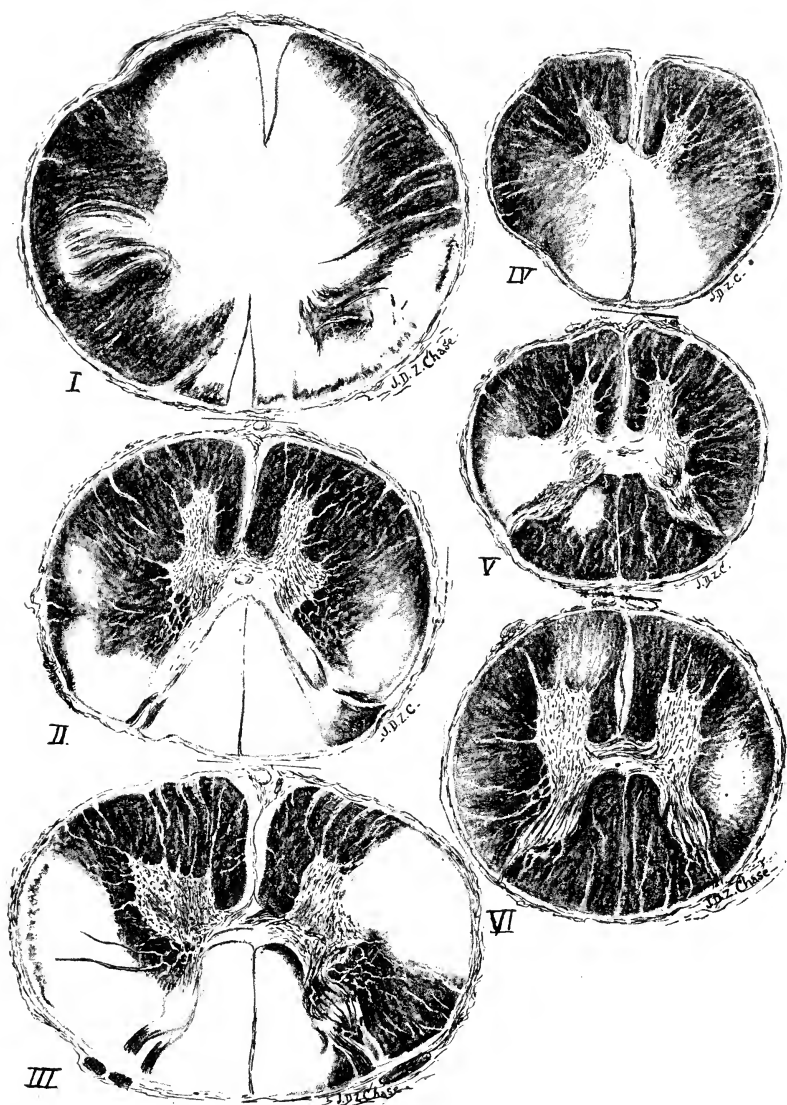
Necropsy revealed the following: The central nervous system—On gross examination isolated areas of sclerosis scattered irregularly over the bulb and cord were visible. These patches varied in color, some being yellow, others gray, and still others of a gelatinous appearance, at times so indistinct as to be overlooked and detected only in the stained specimen. These latter areas were sometimes very extensive. The microscopic

examination of the cord revealed extensive degeneration following in places the type of system degeneration, *i. e.*, confined to the lateral or postero-lateral tracts; at other places affecting the cord irregularly. In the dorsal cord the areas were very numerous and irregular in distribution. (See Figs. iv and v.) Only the ninth and eleventh segments were free, the others having one or more areas of sclerosis. The lumbar enlargement was comparatively free, there being only one small patch of sclerosis in the third lumbar. The medulla oblongata contained several areas of degeneration; one of these extended from the ventral to the dorsal surface; the other areas were irregularly unilateral. The twelfth nucleus was involved, but the cells showed no other change than excessive pigmentation. The pontine lesions consisted of several areas mostly limited to the right side of the pons, although several minute areas were present on the left side. On the right a rather extensive patch extended along the ventricular surface embracing the sixth and seventh nuclei, and involving the inferior surface of the cerebellum. The basal ganglia were free. Several small patches were scattered through the centrum semi-ovale, and very extensive areas were present in the neighborhood of the inferior horn of the lateral ventricle.

The cerebral cortex presents many interesting features. On gross examination of the Müller hardened specimen, extensive areas stained a light yellow without differentiation from the underlying white matter. Under the microscope these degenerated areas contained no tangential fibers. The white substance for several millimeters remained unstained and contained many compound granule cells filled with a pigment staining black with the Weigert method. The glia cells were also markedly enlarged and contained the same pigment. The ganglion cells were very scarce and were very yellow from pigment and rarely shrunken and atrophied. The vessel walls showed evidence of irritation, in some places being thickened, in others surrounded by a reticular network of nucleated cells. The optic nerves and commissure were almost completely destroyed by extensive areas of sclerosis. The right optic nerve contained practically no myelin at all. Two-thirds of the left nerve was likewise affected. The commissure on cross section contained a small crescent of stained fibers around either end of the section. The other cranial nerves were not affected.

We have, in short, a case which, during a course of seven years, presents the clinical picture of a posterior sclerosis; of an ataxic paraplegia (postero-lateral sclerosis); and imme-





The drawings represent the areas of sclerosis in different portions of the spinal cord.

diately before death of a spastic paraplegia; the typical symptoms of multiple sclerosis never at any time being present.

That a disease process so extensive and irregular in its involvement of nervous tissue, so irregular in its etiology and development, should fail to follow any one clinical type is rather to be expected than otherwise. It is not surprising, therefore, that several irregular or atypical forms should be found described. Besides the usual cerebro-spinal form we have the cerebral and the spinal form, the bulbar, the myopathic and optic varieties. We have a still further division into the acute, subacute, and chronic, according to the course of the disease. These different forms run one into the other, but besides the different cases which may be arranged under these various headings there are many which cannot be so classified and are described as atypical forms. The diagnosis in our case was made difficult not only by the entire absence of what are usually considered the typical symptoms of multiple sclerosis, *i. e.*, the intention tremor, scanning speech, nystagmus, loss of mental power, etc.; but also by the course of the disease itself, which, by picking out certain system tracts of the cord at successive intervals, led to the diagnosis by different observers of the disease suggested by the tracts affected. The diagnosis of *tabes dorsalis* made at the onset is easily explained by the pathological examination. A dense and by far the oldest plaque of sclerosis is found in the dorsal region and is almost entirely limited to the posterior columns. (See Fig. iv.) In no other area affected is there such complete destruction of both myelin sheaths and axis cylinders, and in no other area does the glia take on such a wavy and fibrillar appearance on long section as here. A differential diagnosis in this stage would certainly be very difficult. Whether the eye-grounds at this period presented any changes it is impossible to state, but inasmuch as the patient did not complain of even haziness of vision for a year or so later it is probable that no change existed. Granting such to be the case, the absence of pain becomes an important negative symptom, and a possible determining factor between lesions affecting the posterior columns and those in which the roots are also in-

volved. With involvement of the lateral tracts spasticity was added to the other symptoms, and under a different observer ataxic paraplegia (combined system disease) was diagnosed. The lesions in the cervical cord show a much more advanced degree of sclerosis than the dorsal or cerebral lesions, and it was probably these lesions which caused the symptoms. (See Figs. ii and iii.) The lateral and posterior columns are much more likely to be affected in cervical sclerosis than other portions of the cord. We find this to a certain extent true in the cases of others where we have been able to examine the diagrams of the affected cords. The differential diagnosis at this stage would still be very difficult, but might have been suggested by the partial optic atrophy which probably developed at this time. When the case came under our observation the loss of power was so extensive and the spasticity so great that it was impossible to determine any ataxia even had it been present. Unaware of the previous diagnoses, and in the absence of sensory or other localizing symptoms, a provisional symptomatic diagnosis of spastic paraplegia was made. The eye examination would, in all probability, have made a correct diagnosis possible, but was not made on account of lack of proper facilities and because the patient did not complain of any failing vision, and even the night before his death wrote some letters.

The case presents many interesting and instructing features. It suggests the importance of a careful eye-ground examination in that large class of cases too frequently diagnosed spastic paraplegia, meaning, of course, primary degeneration of the motor tracts, and which almost invariably turn out post-mortem to be something else. Attention has already been called to the frequency of ocular symptoms in multiple sclerosis. The changes to be expected are complete or more frequently partial optic atrophy, pallor and loss of definition of the temporal side of the disc, hyperemia of the disc, and rarely optic neuritis. These changes may be the initial symptoms and lead to early diagnosis. Of fifty cases analyzed by Grauck, fifteen revealed changes in the fundus, nineteen with pupillary changes, four of which showed rigidity of the pupil.

Such changes, even when very extensive, may be accompanied, as in our case, with comparatively little disturbance of vision.

That the complete absence of bulbar symptoms (scanning speech, wasting of the tongue, etc.) does not necessarily imply the freedom of the bulb from sclerotic patches is fully proven by the extensive involvement of both pons and medulla in this case. The contention of Erb and Ordenstein that lesions of the pons are the cause of the intention tremor would seem to be disproven by the findings in our case. If such were the case we would expect this symptom in a marked degree on one side on account of the very extensive involvement of the right half of the pons and the almost complete exemption of the left. The widespread involvement of the cerebral cortex by a degenerative process, on the right side spreading over the entire motor area and parietal lobe, and on the left over the temporo-sphenoidal and occipital areas would likewise contravert Greif's assumption that this area (motor) was at fault. The thalamus was entirely normal, and in so far supports the theory of Stephan that thalamic lesions are the cause of intention tremors in this disease. The occurrence of intention tremor in fifty per cent. of the cases, and the comparative frequency of the involvement of the basal ganglia also support this view. The presence of nystagmus in a large per cent. of all cases is interesting in connection with its absence here. The cerebellum, to which this symptom has been attributed, was only slightly involved by a patch of sclerosis extending from the ventricular surface of the pons.

The absence of sensory symptoms was probably accidental; sensation being intact on those occasions when a careful examination for sensation was made. There must necessarily have been sensory changes during the more acute stages of such lesions, *i. e.*, the one in the dorsal region which practically cut off the entire cord, there being only a semi-circle of unaffected tissue around the anterior median fissure. Oppenheim has called attention to the fact that unless the patient has been under observation for a long time and repeated and careful tests made, the sensory phenomena are likely to be overlooked. The sclerotic areas in the cord followed in a

general way two types: Some beginning at the margin and extending inwards, and others beginning near the central canal or at the junction of the gray and white matter (*Grenzschicht*) and extending outwards. In some of the areas of earlier formation the perivascular tissues have a peculiar structure. In those portions of the cord which approach the normal, there being no part where some proliferative change has not taken place, the vessel walls show little if any change. In areas a little further advanced, where the sclerosis is extensive enough to show grossly by the Weigert method, the vessels are surrounded by a reticular tissue which, on closer examination is seen to consist of transparent cells with distinct nuclei. In the early stage of this formation these cells are separated from the surrounding tissue by a distinct wall of cellular tissue. In other portions of the same section this reticular formation extends irregularly into the surrounding white substance. We are led to believe, on account of its structure, its close and constant association with the vessels, its presence in connection with slight thickening of the vessel walls in the early sclerosis, and its absence with marked thickening of the vessel walls in areas of advanced sclerosis, that it is the result of irritation of the vessels by the processes at work in the production of the sclerotic areas. It is also probable that these cells, connective tissue in nature, are productive factors in association with the proliferating glia in the formation of the sclerotic patches. This reticulum has nothing in common with that described by Redlich and others, due to a falling out of the axis cylinder without a concomitant overgrowth of glia. The retention of the axis cylinder in areas of dense sclerosis after complete destruction of the myelin, so commonly seen in this disease, was present to a considerable degree. We think, however, that in certain areas they were too few in number (less than in many cases of tabes) to explain the absence of secondary degeneration by their presence. We have no other explanation for these phenomena.

Such widespread cortical changes we have never seen in multiple sclerosis. On macroscopic examination the Weigert section gave us the impression of a layer of white matter

1-16 of an inch thick, in the position where the tangential fibers lie. This proved to be a layer of Deiters' cells several deep, filled with pigment staining black with hematoxylin. Beneath this an unstained area at least twice as broad as the cortical gray substance indicated the depth to which the process had extended. The presence in those areas of the nerve cells practically intact is what we might have expected from sclerosis of the gray substance of the cord in this and other cases, and explains the lack of mental symptoms, although his inactive life gave little opportunity for judging of the mental acuity which was necessary in his occupation.

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- 139 HERZEN KRANKHEIT UND EPILEPSIE (Relation of Heart Disease to Epilepsy). R. Stintzig (Deutsch. Archiv f. klin. Med., Vol. 66, Dec. 13, 1899, p. 241).

Stintzig reports 2 cases of this condition occurring respectively in a girl of 23 and in a young man of 32; in the former both conditions developed apparently as the result of a severe injury to the breast at the age of 18. From this time there was dyspnea and palpitation of the heart. The treatment of the heart condition seemed to have little effect upon the frequency of the convulsions, nor did they on the other hand produce any alteration apparently in the compensation of the heart. At her death the brain was normal; there was tricupsid mitral obstruction. The second patient developed epilepsy at the age of 22 and the heart disease apparently some time later. He was a chronic alcoholic, and finally died in *status epilepticus* produced by a prolonged debauch. There was mitral obstruction. Stintzig believes that there are three possibilities: either the epilepsy and the heart disease develop as a result of a common cause or they are accidental complications; or one disease may produce or influence the course of the other. He believes that in certain cases some common cause, such as syphilis, may actually produce both conditions. He is skeptical, however, as to whether epilepsy could ever produce heart disease, and thinks it possible that heart disease could only produce epilepsy in a person strongly predisposed to it. The majority of cases in which the complication occurs are youthful individuals. He concludes that the simultaneous occurrence of epilepsy and heart disease is usually accidental; that epilepsy may produce transitory dilatation; that heart disease cannot be the only cause of epilepsy; but may, under certain circumstances perhaps by producing disturbances in the circulation of the brain, contribute to the cause of the epileptic attacks. In some conditions, treatment of the cardiac condition assists the epilepsy; in others the epileptic attacks may be replaced or accompanied by angina pectoris.

SAILER.

A CASE OF MALARIA PRESENTING THE SYMPTOMS OF  
DISSEMINATED SCLEROSIS. WITH NECROPSY.\*

BY WILLIAM G. SPILLER, M.D.

ABSTRACT.

Dr. Spiller reported a case in which the symptoms were: Marked intention tremor of the left upper limb, marked ataxia of the left lower limb, transitory hemiparesis of one side of the body, and later of the other side; headache, vertigo, drowsiness, diplopia, marked vertical nystagmus, distinctly scanning speech, and exaggerated tendon reflexes on the right side. The man died after an attack of severe diarrhea, probably of malarial nature. In the microscopical examination every capillary of the central nervous system was found plugged with pigmented malarial parasites of the estivo-autumnal form. A slight area of sclerosis was found in the outer part of the middle third of the left crusta, and the right crossed pyramidal tract was slightly but distinctly sclerotic. The most probable cause of this slight sclerosis of only one motor tract was probably small hemorrhages of ancient date. This view was confirmed by the numerous small recent hemorrhages and altered blood pigment found within the central nervous system. The only apparent cause of these hemorrhages was the malarial parasite. No areas of disseminated sclerosis were found. The case shows that the symptoms of disseminated sclerosis occurring from malaria are probably the result of vascular alteration of the nerve centers. This seems to be the only case on record in which the symptoms of disseminated sclerosis occurred in malaria and a microscopical examination of the nervous tissues was made.

DISCUSSION.

Dr. Chas. K. Mills said that Dr. Spiller's paper was a valuable contribution. It is possible that he did not understand fully Dr. Spiller's interpretation of the production of the symptoms, but there was one point which seemed to Dr. Mills to have needed more consideration than was given to it. While this was undoubtedly a malarial case, it was a question whether after all the ataxic symptoms, the unilateral symp-

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\*Reported at the meeting of the American Neurological Association, May, 1900.

toms, which led to the diagnosis, were due to toxemia—were due, in other words, to the unilateral intoxication of the nervous centers. Dr. Spiller himself said that probably both halves of the nervous system were affected, and that the sclerosis of one pyramidal tract accounted for the absence of ataxia on the side of the diseased tract.

While the malarial organisms were present in great number, there were also present in considerable number, and in various regions, a number of hemorrhagic lesions of smaller or larger size. The symptoms in a case like this should not necessarily be attributed to toxemia, but we should consider the possibility of their being due to the disseminated hemorrhagic foci.

Dr. F. W. Langdon desired to add a report of a patient in his hospital service, who came in a state of profound coma, for which no cause could be found, as there was no evidence of gross lesion or uremia. The patient simply had an elevation of temperature,  $103$  or  $103\frac{1}{2}$ , and the interne, as is usual in such cases in his service, made a blood examination and found the plasmodia present. The patient was given a full dose of quinine, hypodermically, and the next day was walking around the ward. He had no more coma. His coma was apparently due to malaria.

Dr. W. G. Spiller said he had not had an opportunity of comparing the findings in his case with those of other cases, because no similar case with necropsy was on record. It was possible that small hemorrhages had aided in causing the symptoms, but whatever lesions were found on the left side of the central nervous system were found on the right, with the exception of the moderate sclerosis of the crossed pyramidal tract on the right side of the cord, and the difference in the symptoms seemed to be most satisfactorily explained by that moderate sclerosis.

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140 UEBER DAS GEWICHT DES SCHWERSTEN BIS JETZT BESCHRIEBENEN GEHIRNS (The Weight of the Heaviest Brain Yet Described). G. C. van Walsen (Neurologisches Centralblatt, No. 13, 1899, p. 578).

Heretofore, what was considered the heaviest of all brains was that described by Simms of a young idiot, which weighed 2,400 grammes. Van Walsen describes one that came under his notice which weighed in the recent state 2,850 grammes. It had been possessed by an epileptic idiot, whose two brothers were likewise macrocephalic. The patient learned to walk during his fourth year, but had no paralysis whatever. There were slight spasms in the muscles of the legs. The brain was proportionately enlarged in all its parts. The convolutions were in no wise exceptional in appearance or in position. Upon microscopic examination it was seen that the ganglion cells were relatively wide apart.

BONAR.



NOTE UPON THE OCCURRENCE OF MULTIPLE NEURITIS  
AND BERI-BERI IN ALABAMA.\*

By E. D. BONDURANT, M. D.,

MOBILE, ALABAMA.

PROFESSOR OF NERVOUS AND MENTAL DISEASES IN THE MEDICAL DEPARTMENT  
OF THE UNIVERSITY OF ALABAMA.

An especial interest in peripheral neuritis, engendered of an unusually large experience with it, in both its endemic and other varieties, has led me to make careful search of the literature for reported cases, and numerous inquiries by letter and otherwise, of practicing physicians regarding the occurrence of the disease in this and neighboring States. The result of such inquiry has been the conclusion that while multiple neuritis is, in none of its forms, very common in the Southern United States (even the chronic alcoholic variety being an unusual outcome of intemperance), the disease is met with more frequently than is generally believed, its nature, in many instances, not being recognized by the attending physician.

There occurred in the insane hospital at Tuscaloosa, in 1895 and '96 an outbreak of endemic multiple neuritis, or beri-beri, numbering 71 cases, reported to the State Medical Association, at its meeting in Selma, in April, 1897.<sup>1</sup>

A similar outbreak, on a smaller scale, 25 cases, occurred among the patients of the Arkansas State Insane Hospital, at Little Rock, in the summer of 1895. In 1896, in Green county, Alabama, a number of cases of endemic multiple neuritis occurred in adults, interspersed with cases of acute anterior poliomyelitis in children. These are the only recorded instances of the appearance in the Southern States of endemic multiple neuritis, and the only ones of which I have been able to learn, although a few sporadic cases of similar nature have occurred in Alabama. A circular letter addressed to a large number of the practitioners of the State at the time my report of the Tuscaloosa outbreak was in preparation brought me replies from more than 100 physicians, all of whom stated that

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\*Read by title at the twenty-sixth annual meeting of the American Neurological Association, May, 1900.

<sup>1</sup>New York Medical Journal, November 20-27, 1897.

no case of endemic, malarial or acute neuritis had ever been seen by them. Only two of them had ever seen multiple neuritis of any kind, and their cases, three in number, were all of the chronic alcoholic form. After my paper was read at the meeting of the Association several physicians spoke to me in reference to cases which they had seen and which they thought somewhat resembled those cases of endemic neuritis which I had just described, and I feel sure from the statement of clinical symptoms given by these gentlemen that some of the cases at least were cases of multiple neuritis.

For instance the following:

A young married woman, white, living in a malarial neighborhood, but having had no symptoms of malaria, began complaining of intense neuralgic pains in the legs, and a few days later became so weak that she could not walk. There was tenderness on pressure and some swelling in feet and legs. Joints not involved, no constitutional disturbance. The pains subsided after a week and the edematous swelling in less time. The weakness, considerable, but never amounting to complete paralysis, persisted for several weeks longer, then slowly disappeared, leaving patient as well as before.

A young man, epileptic and very degenerate, began without assignable cause to complain bitterly of pain in his legs, and shortly thereafter became partially paralyzed. He refused to attempt to walk or even to stand, and spent several weeks in bed, complaining much of pain. The attack was at first regarded as hysterical. Later the attending physician became convinced that he had to deal with some form of spinal paralysis. The legs grew smaller, and the patient was nearly helpless for several months, then without any especial treatment he began to improve, and in course of time entirely recovered.

Two other physicians, each of whom had previously written me that he had seen no case of neuritis, subsequently stated to me that they had had cases which resembled those referred to in my paper and which they now regarded as acute multiple neuritis. These cases were as follows:

A young negro man had an acute attack of "biliousness," with fever and gastro-intestinal disturbance for several days. As he recovered from this attack he found that his legs were weak and tender to the touch. The weakness increased rapidly, attaining such degree that he was unable to walk for several weeks. There was very little pain. His mother

thought that he was "conjured." Patient was for a time much distressed about being "paralyzed," but soon began to improve and in a few weeks was entirely well.

A young white woman was for an entire day exposed to cold and wet. On the following day she developed what was at first thought to be "acute rheumatism." She complained of "pain all over," became weak and finally helpless in all four extremities; face, body and limbs were swollen and edematous, and the entire cutaneous surface was exquisitely painful to the touch. The respiration and heart action became rapid, the latter excessively so, and the arteries in the neck pulsated violently. Patient lay helpless for five days, suffering intense pain and dyspnea, and then died of "heart failure." This case was surely a typical acute beri-beri of the "wet" variety.

I have, since 1897, learned of the occurrence of about a dozen cases of multiple neuritis of various kinds in the practice of fellow physicians, and have myself seen and treated eleven well-marked cases (in addition to the cases of beri-beri below referred to). Of these eleven cases, two were syphilitic in origin, two the effect of chronic alcoholism, one probably malarial, one post-typhoid, and five developed without assignable cause. One of them had previously been diagnosed "locomotor ataxia," and three as "rheumatism." All were of gradual and insidious development and pursued a chronic course, and all confirmed the diagnoses by recovering; two still under treatment, improving.

The form of multiple neuritis most common in and about Mobile is beri-beri, cases of this disease being seen every year among the crews of vessels coming into this port. The Norwegians suffer more than do the sailors of any other nationality, and of late years, in consequence of its frequency and severity and the high mortality resulting from it, have learned to dread beri-beri in tropical voyages more than any other disease with which they have to contend. The disease occurs not only on ships coming into Mobile, but in outward-bound vessels as well. So common is it on the sailing ships which load with timber at this and other points on the Gulf that the Norwegian sailors now speak of Mobile, Pensacola, and Ship Island as the "beri-beri ports." One Norwegian captain tells me

that there are only two ports in the world where there is more dread of beri-beri than at the Gulf ports named; these two being Rangoon and Bangkok, E. I., where the ships load with teak wood in much the same manner as they do with pine timber at Mobile. It is noteworthy, however, that the disease is unknown among the inhabitants of the Gulf coast; and careful inquiry has failed to unearth a single case among the Mobile timbermen—the laborers who load the ships with timber—although, as above stated, the crews of these ships often develop beri-beri a few days after leaving port. It would seem that the source of infection is on the ship; so far as I have been able to learn the ships in which beri-beri develops after sailing from Mobile are those upon which beri-beri had previously appeared—ships infected in the East Indies or other tropical ports.

Some of the masters have been inclined to ascribe the disease to the general use of canned food, but most of them now recognize the infectious nature of the malady and think that the poison is developed in the cargoes of wet timber. They also observe that after the disease has once appeared on a ship it is apt to recur in subsequent voyages.

During the past twelve months I have seen and examined seven cases of the disease, all of them giving the typical history and clinical symptoms of beri-beri. Other cases have been treated at the United States Marine Hospital and at the City Hospital. Dr. H. T. Inge, who has a large practice among the sailors of this port, informs me that during the past few years he has treated probably fifty cases of the disease.

I would say in passing that the epidemic form of neuritis seen at Tuscaloosa, in 1896, above referred to, was unquestionably a true beri-beri, paralleling in all of its features the disease as seen among the sea-faring men at Mobile.

SECTION OF THE POSTERIOR SPINAL ROOTS FOR THE RELIEF OF PAIN IN A CASE OF NEURITIS OF THE BRACHIAL PLEXUS; CESSATION OF PAIN IN THE AFFECTED AREA; LATER DEVELOPMENT OF BROWN-SÉQUARD'S PARALYSIS AS A RESULT OF LAMINECTOMY; UNUSUAL DISTRIBUTION OF ROOT ANESTHESIA; LATER PARTIAL RETURN OF SENSIBILITY.\*

BY MORTON PRINCE, M.D.

ABSTRACT.

The important features in this case are mentioned in the title. A man had paralysis of almost the entire left upper limb, as the result of neuritis following an accident. Division of the posterior roots of the fifth, sixth and seventh cervical nerves on the left side was performed on account of intense and persisting pain in the thumb and forefinger and corresponding half of the middle finger, back and front; and of less intense pain extending like a half-bracelet around the back of the wrist, and from the middle point of this bracelet up the middle of the forearm to the olecranon. The operation gave complete relief from this pain in the left upper limb, but caused Brown-Séquard's paralysis, the motor weakness being on the left side. This paralysis was probably due to hemorrhage within the spinal canal or pressure from granulation tissue or callus. Pain, lasting some months, developed in the neck after the operation, and was probably from an injury to a nerve in this part. After the operation the absolute loss of sensation for touch and pain was limited to the whole thumb with its ball, back of the hand, and a strip along the radial aspect of the forearm and arm. The palmar surface of fore and middle fingers was not anesthetic. Dr. Prince believed that the absolutely anesthetic strip along the radial edge of the forearm was in part or wholly due to the division of the fifth root. The sixth root in the hand could not have supplied more than the thumb (front and back) with its ball, though it probably aided in giving sen-

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\*Read at the meeting of the American Neurological Association, May, 1900. To be published in full in *Brain*.

sory fibers to the back of the forefinger. The seventh cervical probably gave a more extensive supply to the back of the hand than usual, but not to the fingers. The remainder of the backs of all four fingers, and nearly the whole of the palm and palmar surfaces of the fingers must have been innervated through the eighth cervical or first thoracic. A remarkable partial restoration of sensation in the anesthetic area was attributed to overlapping nerve supply from adjoining root areas.

#### DISCUSSION.

Dr. A. Meyer said he had mentioned recently to Dr. Prince that of late we are again considerably at sea with regard to the anatomical and clinical correlations of the Brown-Séquard paralysis, since Brown-Séquard had stated, in his last publication on the matter, that he had to change his view completely concerning this subject, because, on cutting the posterior root he had obtained the Brown-Séquard symptom-complex; further, by making a second hemisection lower down, after producing the Brown-Séquard symptoms, he was capable of simply reversing the whole picture in the posterior segments of the body. So the question arose whether it was really necessary to assume that a blood clot was active in the production of Brown-Séquard paralysis. Dr. Meyer had mentioned this to Dr. Prince because he was desirous of knowing whether any of the neurologists present had any experience of their own, experimental or pathological, which would remove again the doubts that Brown-Séquard himself had expressed. It is undoubtedly one of the most difficult questions to examine experimentally, because of the difficulty of determining an anesthesia in animals, and whenever experiments of the sort are made, it is evident that exceedingly accurate microscopical examination with serial sections—not with simple sections taken at random—of the whole region of the wound will be necessary to establish the question whether perhaps disorder of the blood supply, or important hemorrhages during operation, had taken place.

Dr. M. Prince said he had nothing more to say, excepting that he had never seen a laminectomy performed, especially when the dura was opened, without thinking of the possible danger of an artificial blood-clot being formed in the canal, and later pressing on the cord. He had called the attention of the surgeon to this danger. As one watches an operation it seems strange that blood does not gravitate into the canal. It looked

as though this had actually occurred in this case, or else that pressure upon the cord had been caused by granulation tissue. Dr. Meyer suggested the other possibility, a purely physiological explanation, which is interesting. But on the other hand, it well may be that the paralysis which Brown-Séquard noted in his experiments was likewise due to injury from blood clot or other pressure. Perhaps the explanation may not be the same in two cases.

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- 141 UEBER DIE SELBSTWAHRNEHMUNG DER HERDER-KRANKUNGEN DES GEHIRNS DURCH DEN KRAANKEN BEI RINDENBLINDHEIT UND RINDEN-TAUBHEIT (Cortical Blindness and Deafness as Observed and Regarded by the Afflicted Themselves). G. Anton (Archiv. für Psychiatrie und Nervenkrankheiten, xxxii, Heft I, 1899, p. 86).

Circumscribed cortical disease and the resulting functional disturbances are noticed and observed in different degrees by the patients themselves. Anton gives the results of his observations on three cases in point.

Case I. Patient notices neither spoken words nor noises of any kind, reads and writes wrongly, denies being deaf, however, but suffers with hallucinations of hearing and ideas of insanity. The auditory hallucinations point to disease of the temporal lobe. The attitude of the patient as regards his disturbances of hearing is proof that the most central perceptive centers for hearing are disturbed.

Case II. With only a slight disturbance of speech, which the patient herself regarded as abnormal and annoying, there also existed a hemianopsia, of which she took absolutely no notice. Furthermore, there was inability to use her position-sense. She was able to remember objects seen. Upon autopsy there were found symmetrical foci in both occipital lobes, as a result of which the fibers of the optic tract were interrupted in their course. Anton's conclusions as to this case led him to believe that the injuries of the cuneus and the calcarine fissure were not sufficiently extensive to cause a permanent ignorance on the part of the patient of her hemianopsia, and that there must have been still further brain lesions in order that the degeneration of one of the sense functions remained latent to the patient.

Case III. Patient did not understand spoken words and did not respond to noises. There was paraphasia, but she retained the power to read and write quite well. The loss of her power of hearing was not at all recognized by the patient, although she often noticed that the words she uttered were wrong. The autopsy disclosed bilateral softenings in the first and second temporal convolutions and in the lower parietal lobe, as well as secondary degenerations. The auditory nerves and their roots were intact. From these and other cases he concludes that bilateral lesions of the temporal lobe have other results and characteristic symptoms than the mere sum of those seen in unilateral lesions of right and left temporal lobes would have. Complete deafness may result from bilateral lesions of the temporal lobes, while at the same time the patient is entirely unaware of it. This symptom gives us a new diagnostic means to differentiate bilateral temporal disease from peripheral deafness.

JELLIFFE.

## NEW YORK NEUROLOGICAL SOCIETY.

June 5, 1900.

The President, Dr. Frederick Peterson, in the chair.

### EXOPHTHALMIC GOITRE TREATED BY INTESTINAL ANTISEPTICS.

Dr. Mary Putnam Jacobi presented the following case: B. D., aged 22 years, was first seen February 8th, 1899. She had been well until the age of twenty, when she began to work at a shop for making infants' clothing. She worked from eight until five on button-holes and feather stitching. She began to suffer much from constipation, but was relieved by frequent purgation with licorice powder. At the end of a year menstruation ceased, *i. e.*, in July, 1897.

Six months later the eyeballs began to be prominent, and at the same time she had flushes of heat all over the body, palpitations of the heart, headache, weakness, tremor of hands and feet, especially on rising in the morning, and frequent distress in the stomach. In the summer of 1898, constipation was replaced by chronic diarrhea.

In February of this year she had a severe attack of diphtheria, for which she entered Mt. Sinai Hospital. On recovery from this she felt much better for a while.

The thyroid gland was only moderately enlarged and appeared to be slightly larger in the morning at the same time the tremor and diarrhea occurred. Both cardiac sounds were markedly accentuated. No murmurs were heard over the heart, but a systolic murmur was heard over the carotid artery. The pulse was 120; respiration was 24; sphygmogram was regular and rapid.

The patient complained of constant aching in upper thoracic region between the shoulder blades.

An attempt at a complete milk diet rendered the patient very weak and miserable, and increased the tachycardia to a point where the pulse could not be counted.

The urine was examined by Bouchard's method twice this winter, once in February, and again in March. On the first occasion the toxic coefficient was .579, on the second, .712.

The patient had been treated by various nervous and vascular tonics, without material benefit, before she came into the hands of Dr. Jacobi. Various disinfectants were used by Dr. Jacobi at different times for a long period, bismuth salicylate and beta naphthol, associated with a daily flushing of the colon with two quarts of water. Under this treatment, and especially on account of the colon flushings, the diarrhea was



arrested, and the patient grew steadily stronger. Menstruation returned on April 15th, 1899, after an amenorrhea of twenty-two months. A third examination of the urine gave an urotoxic coefficient of 1.426, nearly double the toxicity found in March. For eleven months the pulse remained at 120.

On April 21st, the patient was feeling very weak, with much headache and anorexia, and the urotoxic coefficient was .388, the only time at which it was subnormal, or any other than excessive. On March 27th, after an experiment with thymus gland, the coefficient was 1.662; menstruation occurred three days later. After this it was absent until January, 1900, when it returned, and has returned regularly ever since.

The last urinary examination was made May 22d, 1900, and the coefficient was then nearly normal, being .432.

The pulse remained at 120 or over, with scarcely an exception for eleven months; then, on January 24th, it became 100 and did not change in spite of an attack of diarrhea and insomnia. On the 7th of February, 1900, the medication was changed to salicylate of soda, of which from 60 to 80 grains were given daily. After ten days the pulse was found to be 93, ten days later 92, and on April 4th, 90.

An intercurrent attack of diphtheria treated at home by muriate of iron, had the effect of lowering the pulse to 90.

Dr. E. D. Fisher said that this patient had a very vascular condition of the thyroid gland. He did not absolutely agree with Dr. Jacobi in the use of intestinal antiseptics. He had found the use of strophanthin for long periods successful. That treatment might be carried out in a case of this kind.

Dr. Wm. H. Porter said he had observed two cases of a similar nature which were treated by antiseptics of the alimentary canal, to prevent toxic infection, with better results than had been obtained under other treatment for that class of cases.

Dr. Wm. H. Caswell had found that 15 grains of glycerophosphate of soda, given three times a day, had been useful in a case of this kind. The pulse had fallen from 130 or 140 to 84 or 88, and remained low for the last two months. All constitutional symptoms were very much improved, although the exophthalmus had not disappeared, nor was there any decrease in the size of the thyroid. The patient was still doing well.

Dr. Jacobi said that she had paid no systematic attention to this question of the urotoxic coefficient. She had noticed that when the urotoxic coefficient was in excess of normal, the patient was better. One time it fell below normal, and she was decidedly weaker with symptoms of more constitutional poisoning, confirming the idea that poison circulating in the blood and excess of urine toxicity coexisted with the effort of the organism to throw it out. When the toxicity was high, the patient was better; when subnormal, the patient was worse.

## NON-INFLAMMATORY, CONGENITAL HYDROCEPHALUS.

This case was presented by Dr. Joseph Fraenkel. The patient was a child 26 months old, born at full time in normal labor. The father was 36, mother was 30, and both parents were Russian Hebrews. Three children were living and well. The mother had had one miscarriage prior to the birth of the child exhibited. Immediately after birth, this child was noticed to be abnormal. It did not hold its head up, and was generally backward. The symptoms have become more accentuated. It has never walked, talked, or shown any signs of mental vigor. Obstinate constipation has existed since birth. There are no evidences on the skeleton pointing to rachitis. The upper and lower extremities are permanently contractured, but the contractures can be overcome and the reflexes are present and not exaggerated. No ankylosis exists. The child has had three or four general convulsions. After the last one reported, the voice was lost for three or four days. Sensation, in so far as it can be examined, is normal. The head is somewhat large, 46 centimeters in circumference, though not so large as in many hydrocephalic infants. The fontanelles are not closed. The child was treated medically, but objective examination showed no improvement. Dr. Fraenkel thought that the case was one of hydrocephalus, although it was atypical.

Dr. Fisher agreed with Dr. Fraenkel's diagnosis of non-inflammatory congenital hydrocephalus in this case.

Dr. Fraenkel said that in cases of hydrocephalus he had seen, he had not noticed this constant rigidity and throwing back of the head. The head was comparatively small in view of the severity of the symptoms.

Dr. Jacobi thought there might be a correlation between the small head and the severity of the symptoms. For some reason the skull had not yielded, causing greater intracranial pressure than in ordinary cases where the skull is large and necessarily the pressure less.

Dr. Lewis A. Conner suggested that the question of increased intracranial pressure could be determined by lumbar puncture and the use of the manometer. A manometer may be simply improvised by using a bent glass tube with a short horizontal and a long perpendicular arm, and attaching it by a piece of rubber tubing to the canula.

Dr. Peterson said that the fontanelles being open would show that there was not unusually great intracranial pressure. The case did not look like one of congenital hydrocephalus to him. In that disease there should be an unusual growth of the head. He had seen similar rigidity resulting from basilar hemorrhage occurring during labor.

Dr. Fraenkel said that the labor was normal.

## MULTIPLE NEURITIS FOLLOWING PNEUMONIA.

A case of this character was presented by Dr. Conner. He showed the patient because of the unusual association of neuritis with another disease. The man,

41 years of age, came to the Hudson Street Hospital on the 7th of November, with lobar pneumonia of five days' standing, typical history and physical signs. The consolidation involved the left upper and lower lobes, and ran a normal course, but instead of disappearing at the end of the 7th or 9th day, the temperature dropped and the consolidation remained, presenting the ordinary picture of delayed resolution, lasting two weeks beyond the allotted time. During that time the patient gave no evidence of any other trouble. Just at the end of this period of consolidation, three weeks from the beginning of the attack of pneumonia, his legs were noticed to be weak, and he had some discomfort in moving them, and the extensors of the left arm were paretic. In the course of a week the following condition developed:

Extensive, almost complete paresis of the extensors of the leg, of the tibialis and peroneal muscles, and of the extensors of the left arm, there was also slight loss of power in the right arm. No anesthesia was found. Some numbness existed in the tips of the fingers, and an area of hyperesthesia was present over the crest of the ilium on each side, but there was almost no pain except girdle sensation. Tenderness over the calves of the legs and arms was very slight. The diaphragm did not functionate properly, and in about a week from the beginning of the symptoms of paralysis, it ceased entirely to functionate in either forced or natural respiration. The disease was a severe multiple neuritis, which ran a course of five weeks before any improvement could be noticed. The patient can now walk about, and has recovered very largely the use of his left hand. It was six weeks from the time of the disappearance of function in the diaphragm before contraction in that muscle could again be recognized. So severe a neuritis is very unusual in lobar pneumonia. It is recognized as a possible complication, but an unusual one, and in the cases in which Dr. Conner had found it, the neuritis had been local, or else of a mild type. As to frequency of involvement of the diaphragm in neuritis, he had the impression that it is not common.

He neglected to say that the man's history was good in respect to syphilis or tuberculosis, but he had been accustomed to drink three or four glasses of beer or ale daily, and two or three times a week, a glass of whisky.

Dr. R. H. Cunningham said that in alcoholic neuritis the vagus is invariably affected several days before the diaphragm is involved. He asked what was the state of the pulse during the attack. Dr. Conner replied that the pulse remained normal.

Dr. Jacobi asked whether it was supposed that there was any extension of the inflammation from the lung to the diaphragm by contiguity, affecting in this way the phrenic nerves.

Dr. Conner believed that the neuritis was the result of toxemia.

Dr. Peterson said that Dr. Conner's case was quite unusual and certainly was one of simple multiple neuritis following pneumonia. The moderate alcoholism might not have been sufficient to produce neuritis, but the added toxic condition of the blood caused by the pneumonia was sufficient to bring it about.

#### PROGRESSIVE MUSCULAR DYSTROPHY.

Dr. Caswell presented the following case: A young woman, 21 years of age, came to the Vanderbilt clinic seeking treatment for weakness in the hands. She is a maker of artificial flowers. Five or six months ago her hands began to be weak, and she could not work so easily as before. He found upon investigation that from the time she was a young girl she had had difficulty in closing her eyes, and she did not smile and use her lips and mouth as other children did. She was not conscious of this, and had complained only for the past few months that her hands failed at work. He examined her and found a case which, in almost every particular, conformed to the type of primary progressive muscular dystrophy described by Landouzy and Dejerine—beginning in the face without hypertrophy, and the atrophy showing later in the upper extremities. The lower extremities were fairly well developed for a thin person. The arms from the elbows to the fingers were not atrophied, but from the elbows to the shoulders there was marked atrophy on each side. The pectoral muscles about the triangles of the neck were decidedly wasted, the scapulæ were winged, and the face was of the myopathic type.

Dr. Caswell said that the onset of this disease is very insidious, and until the patient notices something which interferes with daily employment or manual labor, he does not seek treatment.

Dr. Fraenkel knew of a case which had existed for a number of years. The disease had been stationary for seven or eight years.

Dr. Caswell said that a case of this kind at the Vanderbilt clinic had progressed very slowly during the past five or six years. The changes in the face were very slight, but were gradually becoming more noticeable.

Dr. Peterson considered the Landouzy-Dejerine type very rare up to two years ago, rarer than the Erb juvenile form, but within the past two years he had seen four perfectly typical cases. The history was usually indefinite except as to the eyes. The patients have never remembered being able to close the eyes. This led him to suggest that some cases, occasionally seen, of bilateral lagophthalmos, might be muscular dystrophy of the Landouzy-Dejerine type. Several years ago Dr. Peterson had thought that this form was a variation of the Erb juvenile type, but now he regarded it as a different syndrome with a different pathological basis.

## PROGRESSIVE MUSCULAR DYSTROPHY.

The case was presented by Dr. Leopold Stieglitz. The patient, a man, 33 years of age, born in Germany, came to this country four years ago. His father died of heart disease; his mother is living. There is no similar trouble in the one brother and the four sisters of the patient. He cannot state exactly when the present trouble began. He has noticed for some years weakness in the legs, with inability to lift them properly from the ground, causing difficulty in walking, and that they tire easily. This condition became worse. Dr. Stieglitz saw the patient four years ago at Mt. Sinai Dispensary. There was then a profound atrophy of the peroneal group of muscles in both legs. There was no disturbance of sensation, and but slight signs of reaction of degeneration. The case was entered as one of the peroneal type of progressive muscular atrophy. Last February the patient returned to the dispensary complaining of weakness in the arms. He was examined, and a very pronounced and typical condition of progressive muscular dystrophy of the body was found in addition to the condition in the legs. The atrophy involved also the gastrocnemius muscle of the calf, and to a certain extent, the muscles of the thighs. On both sides there was atrophy of the pectorals, hypertrophy of the deltoids, and wasting away of the upper arms, with typical drop-foot and atrophy of the lower extremities. The diagnosis was the peroneal and juvenile types of progressive muscular atrophy or dystrophy. The electrical reactions, however, did not correspond to those one would expect in a case of this kind. They were normal in the upper extremities, and absent entirely in the lower. The patient had winged scapulae, protruding abdomen, kyphosis of the back; all indicative of progressive muscular dystrophy.

Dr. Fisher said that there is a finely drawn distinction between the juvenile and peroneal types of atrophy. From the conjunction of the two in the same patient, it would seem that they might belong to the same family.

Dr. Fraenkel said that the three types of dystrophy are shown in this patient. The remarkable feature is the absence of any hereditary factors. This condition is unusual at so late an age.

Dr. Peterson agreed with Dr. Stieglitz that the case was of the juvenile type. He thought there was a marked distinction between the juvenile and the Charcot-Marie-Tooth forms. The two syndromes are not alike. One peculiarity of the latter is that the legs below the knees are affected; here we have a case of the Erb juvenile form in which the thighs are affected. The pathology is the same in this case. He did not see why, in the Erb juvenile form, we do not have other muscles involved besides the shoulder girdle.

Dr. Stieglitz said the unusual feature was the commencement of the disease in the lower extremities. He could find nothing that ex-

actly corresponded with this case. The involvement of the lower legs is not so unusual, but in this case simply the muscles below the thigh were involved. Atrophy of the muscles of the thigh is common in progressive muscular dystrophy. He thought we should distinguish between the Charcot-Marie-Tooth and the Landouzy-Dejerine type on account of the difference in pathology; one is a disease of the nerve, while the other is pure myotrophia. On that account we should not try to connect the two syndromes. Unless the family history be carefully investigated, the disease might be hereditary without being known to be so from the statements of the patient.

#### HEREDITARY CEREBELLAR ATAXIA.

Dr. M. Mailhouse presented a patient, F. F., who was admitted to the New Haven Hospital, January 13, 1900, with the following history: Age, 32; single; birthplace, United States; occupation, postal clerk; father and mother living and well; has five sisters living, and all in good health. One sister died of pulmonary tuberculosis, and another died in infancy; mother's mother also died of pulmonary tuberculosis. A maternal aunt is at present an inmate of the Connecticut Asylum for the Insane. Mother and father have had muscular rheumatism. The patient had scarlet fever and diphtheria at fifteen years of age. When a boy he had rheumatism of mild degree. In 1887 had sunstroke. No syphilis, alcoholism or tuberculosis. The history taken on admission to the hospital states that the patient first noticed in 1887 a dragging of the toes on walking, and that the shoes curled up and were worn at the toes. He did not walk with a stamping gait, and could direct the feet properly. The legs did not cross in walking. On closing the eyes while standing he was unable to keep his balance. Had no pain in the legs but considerable backache. Then the hands began to tremble (1888) and the legs shook on walking. These symptoms increased so that he had to use two canes in walking in 1888; in 1889 he used a crutch and one cane, and in 1890, two crutches. Had lost 57 pounds in weight within the past ten years. Has headaches off and on, but they are not severe. He states that when twenty-one years of age, he noticed an inability to run; he could walk, but when he ran "his legs got mixed up as though they were playing cross-tag with one another." For the past four years erections have been incomplete. At one time he had frequent urination.

He is a man above medium height, of good frame, but somewhat emaciated. As he stands supported by two crutches, the nodding tremor of the head, which is aggravated by walking, is very noticeable.

There is marked asymmetry of the head, a flattening of the occiput, and pronounced sloping of the forehead, with con-

siderable flattening over left parietal region. There is some facial asymmetry also, as is evidenced by a greater prominence of the right supraorbital arch than of the left. The nose is directed toward the right, and the left palpebral fissure is smaller than the right. The gait is markedly ataxic, but he is able to get about fairly well on a level with the aid of crutches, but loses his balance readily on attempting to ascend a step. There is absolutely no muscular weakness in his legs. The knee-jerks are present, but not exaggerated. There is no ankle-clonus. The Romberg symptom is present, and he is supported with difficulty when he stands with closed eyes. There is no impairment of any form of sensation. The movements are performed quickly, and are jerky. The ataxia of the upper extremities is much less evident than in the lower. The speech is noticeably affected, but changes, being at times scanning or staccato, and at other times characterized by indecision and unsteadiness in the muscles of vocal expression; it is decidedly an ataxic speech.

The pupils react neither to light nor during accommodation. There is marked lateral nystagmus, and on looking upward a rotatory nystagmus; when the eyes are at rest there is no oscillation. There are no contractures and no scoliosis. The grasp of the hands is strong and the muscles of the arms show no loss of power. The pectoral muscles are wasted, otherwise no atrophies are present. There is no optic atrophy and no limitation of the visual fields, but he is myopic, requiring a lens of seven diopters.

The superficial reflexes are present, the plantar being very active. Urine presents no abnormality. The handwriting is like that of a tabetic.

A review of the symptoms reveals the marked ataxia of the whole muscular system without any paralytic phenomena. The case resembles Friedreich's ataxia in its mode of development and general appearance, but differs from the typical disease in its later onset, in retaining the knee-jerks, in the late and mild affection of the arms, in the loss of the ocular reflexes, and in the absence of contractures or paralysis. The man has been afflicted twelve years, and there is no paralysis or contracture present. The question might arise as to the case being a multiple sclerosis, but this affection could be ruled out by the mode of onset, by the normal reflexes and sensory phenomena, and the absence of intention tremor. Furthermore the long duration of the illness without any paralyses, ocular or corporal, militated against such a diagnosis.

Dr. Caswell said that in the form of ataxia described, the fundus oculi is generally affected and the knee-jerks are increased; in this way discrimination is made between cerebellar and ordinary ataxia.

Dr. Fisher thought that the sunstroke may have had something to do with the symptoms. He remembered a case of sunstroke followed by symptoms of this type, resembling multiple sclerosis, not conforming strictly to it. Taking that as an etiological factor, we have ordinarily some meningitis, some change affecting the cortex of the brain and producing ataxic symptoms; we see ataxia, inco-ordination of the muscles, spastic condition of the patient,—a degenerative condition secondary to some such lesion as might be caused by the effects of the sun.

Dr. Fraenkel thought that there was a great difference between cerebellar ataxia and ataxia produced by disease of any other parts of the co-ordinatory system.

Dr. Peterson said he had examined the man before the meeting and thought the diagnosis lay between multiple sclerosis and cerebellar hereditary ataxia. He was inclined to think Dr. Mailhouse was right in his diagnosis of cerebellar hereditary ataxia on account of the pupillary condition; no reaction of light,—and because he had more of an ataxia than tremor of the ordinary multiple sclerosis type.

Dr. Wm. B. Pritchard asked whether there were anything of note in the subjective history of this case as regards vision, hearing, or persistence of vertigo or headaches that added to the data.

Dr. Mailhouse replied that there were no subjective symptoms at all, except backache and difficulty of manipulating the legs. He was strongly myopic without optical strain.

#### RAYNAUD'S DISEASE OF A GANGRENOUS TYPE.

Dr. Wm. H. Porter presented this case: The man came to this country some years ago; for a year or so was a hod carrier, and then went into an iron foundry where he has worked since. Has a very pronounced alcoholic habit, and uses tobacco to a considerable extent. In the middle of April he noticed that the fingers of both hands were very white and icy cold. This lasted for two weeks and was followed by superficial gangrene, discoloration and blackness of the four fingers of both hands, a little discoloration of the left ear, and a spot on the opposite ear. This condition lasted for ten days before he came to the hospital. He suffered intolerable pain and got very little sleep. The urine showed slight traces of albumin, a decrease in urea and pronounced amount of indican.

The patient had a very high pulse and he was given doses of nitroglycerine with strychnine and caffeine to improve the circulation. Lead and opium dressing was applied and gave relief.



## Periscope.

### CLINICAL NEUROLOGY.

- 142 UEBER DIE BEZIEHUNGEN EPILEPTISCHER ANFÄLLE ZUR HARNSAUER-AUSSCHIEDUNG (On the Relations Subsisting Between Epileptic Attacks and the Excretion of Uric Acid). Caro (Deutsche med. Woch., May 10, 1900).

Caro first recapitulates the work of his predecessors in this direction. In 1896 Haig noted that before epileptic attacks there was a diminution in the excretion of uric acid. In 1897 Kieman found that, generally speaking, the amount of uric acid in the blood of epileptics was increased, while Charon and Briche found that in certain cases there was a connection between the alkalinity of the blood and severe epileptic attacks; with fewer and milder attacks there was increased alkalescence of blood, and vice versa. Recently Krainsky has made some important studies; he found 24 to 48 hours before an attack, a regular diminution of acid in the urine, so regular, in fact, as to have a prognostic value. Krainsky regards the diminished amount of acid as due to a toxic action. This poisonous substance causes the convulsion; and is in turn destroyed by it. The blood of epileptics is toxic to animals at or just before the attack. These findings confirm the results given by others. Krainsky's supposition was that the toxic agent is ammonium carbamate, with free ammonia in the blood.

Caro gives a curve which shows the percentage of uric acid and its relation to several epileptic attacks occurring within a month. Epilepsy must be conceived at the present time as a result of intoxication; all other convulsive maladies are of this nature, uremia, eclampsia, convulsions in ordinary poisoning, the convulsions of children attacked by acute infectious diseases. Even the presence of focal disease in the brain does not wholly invalidate this conclusion; for a symptom complex may be caused by different affections.

CLARK.

- 143 EPILEPSY. W. P. Spratling (Buffalo Med. Jour., June, 1900).

In this paper Spratling says that the disease is found most frequently (1) during early infancy and dentition; (2) during the period covered by the seventh and eighth years, and (3) during the marked transitional period of puberty. Among the causes are: (1) direct transmission from parent to child; (2) accidents at birth; (3) intense pathological processes that should be physiological, such as difficult dentition, especially in rachitic or scrofulous children; (4) indigestion and malassimilation; (5) accidents of early life, and (6) the grave physiological disturbances that come at puberty. For prognostic purposes it is essential to divide epilepsy into that form due to some physical defect or deformity and into the true form of epilepsy in which no direct cause is apparent and in which the physician can do far less good. Treatment may be in institutions or at home; in the latter case, too much parental sympathy, the too liberal use of medicines, especially of the proprietary kind, and improper food, often defeat the object in view. It is important that patients should have meat but once a day, and that at noon and only in small quantities; they should have nothing

fried in grease and no pies or other pastry. They must eat largely of cereals, milk, fruits, eggs and butter. Of drugs, the fluid extract of horse-nettle berries has given satisfaction, since it does not impair the functions of the digestive tract. Simulo, also, has given excellent results. A combination of bromide, chloral and morphine is often of service in aborting an expected seizure preceded by a long aura. Great stress is to be laid upon the systematic exercise of all the muscles of the patient's body, such work as can best be done being prescribed after a thorough physical examination.

JELLIFFE.

144 THE ETIOLOGY AND PATHOLOGY OF MAJOR EPILEPSY. William House (Phila. Med. Jour., Vol. 5, 1900, p. 691).

In this paper the writer reviews briefly the various theories advanced by different observers as to the causation and pathology of epilepsy, and, believing that "like causes produce like effects," compares the symptoms of epilepsy with those of other diseases which produce or are accompanied by convulsive seizures resembling more or less the seizures of epilepsy. These diseases are hysteria, tetany, infantile, puerperal and uremic eclampsia, alcoholism, cerebral hemorrhage, and the apoplectic and epileptiform convulsions of general paresis. The pathology of the three latter conditions is well known, and in certain respects the lesions are of a similar nature. The symptoms of an epileptic seizure, of an epileptiform paretic convulsion, of an alcoholic convulsion, and of cerebral hemorrhage present a marked similarity, so much so that sometimes it is difficult to make a differential diagnosis. Symptoms of cerebral pressure are present in these three conditions and also in epilepsy. In the brain of an alcoholic there is an excessive quantity of cerebrospinal fluid, the ventricles are distended, the brain substance drips with fluid, and the membranes are dropsical. This is called the "wet brain." In general paresis the ventricles are distended with fluid, there is an increased quantity of fluid in the subdural space, and the whole brain is surrounded with an excessive quantity of turbid cerebrospinal fluid. In both these conditions the excessive fluid seems to the writer to be the logical cause of the pressure symptoms recognized in convulsions from these diseases.

The writer has witnessed the autopsies of five cases of *status epilepticus*, and in each instance there was found an excessive quantity of cerebrospinal fluid.

Arguing from analogy, House is forced to the conviction that this increase in the quantity of cerebrospinal fluid must bear a causal relation to the convulsions of epilepsy. According to physiologic findings, if the cerebrospinal fluid be suddenly withdrawn convulsions may ensue; if rapidly increased coma may be produced.

From the observation of over 200 epileptics, and from the comparison of their symptoms with those of 60 alcoholics and those of a large number of paretics, from the findings in the above five autopsies, and from an analysis of recent literature, he concludes that:

(1) There is no record of pathologic findings which logically explain the symptoms of epilepsy.

(2) An increase of cerebrospinal fluid would readily account for the seizures. In many instances it is analogous to the marked increase of fluid in the crania of alcoholics and paretics, and is not dissimilar in clinical effects to the more localized lesions of hemorrhage or abscess.

(3) This fluid, physiologically subject to more or less variation in quantity from day to day, is fully capable of pathologic increase, and from analogy must bear exciting relation to the convulsion.

(4) Its increase is probably gradual, and to this we may ascribe the *auræ*.

(5) Its absorption probably begins with the third stage of the convulsion (relaxation and coma), and if this fails, repeated convulsions (*status epilepticus*) ensue.

(6) Its superabundance may be due to lymphatic spasm, or to marked disturbance of equilibrium between lymphatic and general circulatory activity, which may be favored by heredity, toxemia, or any of the recognized predisposing causes.

(7) This creed applies to the so-called idiopathic epilepsy, as distinguished from the convulsion of the Jacksonian epilepsy, although even in such cases this condition will help to explain some otherwise unexplained symptoms.

BONAR.

145 ZUR KENNTNISS DER PROGRESSIVEN MUSKELATROPHIE (On Progressive Muscular Atrophy). Friedel Pick (Deutsche Zeitschrift für Nervenheilkunde, Vol. 17, Nos. 1 and 2, p. 1).

A man, 52 years old, said that he had been healthy until two years previously, when he suffered from articular pain in the lower limbs, and had weakness of the upper and lower extremities and of the neck musculature. When he was examined the atrophy was quite marked, especially in the sterno-cleido-mastoid muscles. The thenar and hypothenar eminences were also much wasted. Sensation was not disturbed, reflexes were feeble, and there were no fibrillary tremors and no reaction of degeneration. A year after this examination the speech was found to be much disturbed, and fibrillary tremors of the tongue were observed. The case was believed to be one of spinal muscular atrophy. A necropsy was obtained and the spinal cord, brain and peripheral nerves were found to be intact, except the spinal accessory nerve. "Simple" atrophy with lipomatosis was found in the muscles. The post-mortem findings showed that the case was one of muscular dystrophy.

Weakness and wasting following articular pain in the lower extremities seemed to indicate that the case was one of polyneuritis, but against this diagnosis was the absence of sensory disturbances and of reaction of degeneration, although quantitative changes were observed. The case could hardly be considered as one of neurotic muscular atrophy. The short duration and the mode of commencement of the atrophy, the age of the patient, the striking atrophy of the small muscles of the hand, the escape of the trapezius, and the disturbances of speech, seemed to make the diagnosis of muscular dystrophy improbable, and that of spinal muscular atrophy more probable.

Pick discusses the significance of the muscle spindles and the Renault's bodies in nerves. The degeneration of the spinal accessory nerve was believed to be secondary to that of the atrophy of the sterno-cleido-mastoid muscle.

The age of the patient—50 years—at the time the muscular dystrophy began was noteworthy. The appearance of this disease after the age of forty seems to have been observed only by Landouzy and Dejerine, Erb, and Linsmayer. The involvement of the small muscles of the hand in the early stages of dystrophy, as in this case, has been observed only a few times. The intense atrophy of the sterno-cleido-mastoid muscle, with relative integrity of the trapezius, was also unusual. Pick refers to much of the literature on the subject of muscular dystrophy, and comes to the conclusion that muscular dystrophy should be regarded as a primary myopathy.

SPILLER.

- 146 PARALYSIE PSEUDO HYPERTROPHIQUE DU DUCHENNE CHEZ UNE FEMME ADULTE, CONSEQUENTE A UNE FIÈVRE TYPHOÏDE (Pseudo-hypertrophic Paralysis (Duchenne) in an Adult Female, Following an Attack of Typhoid Fever). M. Jossierand (Lyon Médical, June 17, 1900).

A case is here reported of a woman of 26, who at 23 was treated for "fièvre typhoid, myositis legere." The typhoid was treated by the Brand method, and during the baths the calf and arm muscles became swollen, reddened and tender. When the patient was discharged she was in a very feeble condition. The weakness in the legs progressed and she was unable to walk any distance or ascend a flight of stairs. Eighteen months after the typhoid the leg muscles began to hypertrophy. The gait, elevation from a lying posture, reflexes, etc., corresponded to the pseudo-hypertrophy seen in children. He differentiates this condition from the typhoid myositis, which becomes chronic, by the general distribution, progressive course and pseudo-hypertrophy.

McCARTHY.

- 147 INTERMITTIRENDE CLAUDICATION (Contribution to the Knowledge of Intermittent Claudication). Karl Grassmann (Deutsch. Archiv. f. klin. Med., Vol. 66, Dec. 13, 1899, p. 500.)

The author, after a brief discussion of this condition, chiefly historical, and after calling attention to a similar condition that occurs in horses, reports the case of a man 60 years of age, who had suffered from luetic infection, was passionately devoted to riding, and who used alcohol and tobacco to excess. He had first a severe attack of pain in the left leg while fishing; then another attack characterized by the symptoms of thrombosis of the femoral artery, in which the thrombus could be distinctly felt. After this the leg atrophied. A year later there was a similar attack in the right leg. From this time, both limbs were small, and the patient at every hundred steps was attacked by severe pain in the legs, lasting several minutes, and only relieved by rest. During the intervals the feet often felt cold, and were cyanosed, particularly if they were allowed to hang down for any length of time. There was marked hyperhydrosis in the feet. During the treatment, one of the toenails fell off. Treatment consisted of mercury, potassium iodide, and careful massage. It caused considerable improvement, but not complete cure. The case is interesting on account of the presence of the paresthesia, the trophic disturbances, and the history of thrombosis of the arteries. There was arterial sclerosis, which must be made accountable for part of the symptoms, but it is difficult to understand why, if it bears such an important etiological relation to the condition, intermittent claudication is not more frequent in persons with arterial sclerosis, or in those in whom the arteries have been tied.

SAILER.

- 148 AMAUROTIC FAMILY IDIOCY. J. H. Claiborne, Jr.

At a recent meeting of the New York County Medical Society, Dr. J. H. Claiborne, Jr., said that attention was first called to a set of symptoms resembling those caused by embolism of the central retinal artery. The optic disk became atrophic and the other well-known changes occurred. Three similar cases were noted in the same family. All of the children died before they were two years of age. Within three years after these first cases were reported five other cases came under the observation of various ophthalmologists. Then Sachs, of New York, described certain cases that had occurred in his practice as a phase of idiocy. He saw four cases in two families. About a year ago

Carter collected the cases reported to date. They were twenty-nine in number. The development of the condition seems to be about as follows: The child seems perfectly normal at birth. It continues to be normal for three weeks to a month, and then ceases to take any interest in its surroundings. It is often sleepless; it rolls its eyes more or less continuously, and is generally restless. When it should have strength enough to sit up, it fails to do so. It slides down on its back instead of sitting up, but does not fall forward or sideways. Vermicular motions in the fingers somewhat resembling the movements of athetosis have been noted. The tendency to somnolency becomes more marked. Hydrocephalus has been known to develop. Diplegia exists at times, and a spastic condition of the lower limbs has been noted. The electric reactions are normal. At the end of a year the child is totally blind, the optic nerve is of a dead-white color, more or less marked idiocy has asserted itself, and distinct palsy is present. Marasmus usually closes the scene before the end of the second year. The marked differential sign of the disease is the occurrence of idiocy in connection with blindness.

In a recent case seen by Dr. Claiborne the child was normal at birth and remained so up to the ninth month. Then an external squint was noted in the right eye. After this ptosis occurred in this eye. Then a similar train of symptoms developed in the other eye. The family history showed that the father died of tuberculosis. There was no syphilitic history, yet mercurial inunctions were tried on general principles, and after a while the ptosis disappeared and the squint was modified. Marasmus developed, however, and the intelligence decreased very markedly. As the malnutrition advanced the eye symptoms recurred, and finally death from exhaustion ensued. At the autopsy a tubercular tumor of the corpora quadrigemina was found. There was also generalized tuberculosis. Tuberculous nodules were found in the lungs, spleen, and the bronchial glands. Curiously enough this case was not of Hebrew origin. All the subjects of amaurotic family idiocy up to this one were Hebraic. In another case under Dr. Claiborne's care the subject was also not of the Hebrew race. The existence of the tubercle at the base of the brain is extremely interesting. It is possible that in most of the cases some such lesion has been present. At least it is suggestive to find that a tubercle can cause a set of symptoms that resemble amaurotic family idiocy so much.

The differential diagnosis is not difficult as a rule. Freidreich and Marie's ataxia in their hereditary form may be confounded. The pathognomonic sign is a condition of the fundus in the amaurotic family idiocy. The fact that death takes place from marasmus probably points to the tuberculous character of the affection. This characteristic has been missed so far because too much attention has been directed to the central nervous system, especially to the cortex. The suggested pathology of the disease up to this has been some disturbance of the cortex, while the ganglia at the base of the brain and the medulla were considered to be normal. Amaurotic family idiocy is evidently not so rare as has been thought. Seven cases have been under treatment at the Vanderbilt clinic. All of these were Polish Jews, and there was no history of phthisis or of consanguinity in the parents. JELLIFFE.

- 149 PROGNOSE UND THERAPIE GEHIRN LUES (The Prognosis and Therapeutics of Syphilis of the Brain). v. Hösslin (Deutsch. Archiv. f. klin. Med., Dec. 13, 1899, Vol. 66, p. 281).

v. Hösslin reports 11 cases with 2 deaths. The symptoms were various, some of the patients having attacks resembling epilepsy;

others having attacks resembling apoplexy and hemiplegia, and one patient, after apparent complete cure, developed parietic dementia a year later. Several of the others had slight relapses, which were readily amenable to treatment. In one very curious case the patient had multiple vanishing tumors of the scalp, that when excised were found to be due to syphilitic caries of the skull. The treatment consisted exclusively of inunctions of blue mass, potassium iodide by the mouth, and hot air baths. The latter were given at a temperature of about 120°. v. Hösslin calls attention to the great importance of feeding the patients in a state of coma through the esophageal tube. SAILER.

### PATHOLOGY.

- 150 SUR UN CAS DE SOMMEIL PROLONGÉ PENDANT SEPT MOIS PAR TUMEUR DE L'HYPHYPHSE (A Case of Prolonged Sleep, of Seven Months' Duration, Caused by Tumor of the Hypophysis). F. Soca (Nouvelle Iconographie de la Salpêtrière, 13th Year, No. 2, March-April, 1900, p. 101).

A case of a woman 18 years old. No hereditary history, no syphilis or alcohol. Following an attack of sudden unconsciousness, symptoms of blindness, headache, and difficulty in walking developed. Vision rapidly disappeared. An ophthalmological examination showed almost no sight; a double optic atrophy, more pronounced on the left side, dilated pupils, not reacting to light; no ocular paralysis. Walking became impossible, and incessant vomiting, cerebral in type, developed. The most striking symptom after this was the tendency to sleep. Sleep seemed natural in every way except in point of duration. She could be roused with difficulty to take nourishment, but would immediately fall asleep again. This condition lasted seven months, during the greater part of which the patient slept. During her stay in the hospital pain and vomiting ceased. Patient died of broncho-pneumonia. Autopsy: The frontal and occipital poles of the brain were easily detached, but the base was held firmly to the skull by a neoplasm, which held the central part of the base of the brain to the dura mater. The tumor covered the sella turcica, situated directly in the tract of the first pair of cranial nerves. The olfactory peduncles were somewhat spread apart; they did not appear changed otherwise. The optic nerves, from the most anterior portion of the optic tract to the commissure, were adherent to the tumor, or rather so much a part of the tumor that they could not be distinguished from it. The tumor further extended posteriorly on both sides, involving the third and fourth pair, but no adhesions were present, and these nerves appeared in no way affected by the mass. The tumor was sarcomatous in type, possibly developing at the expense of the hypophysis. In discussing the clinical aspects of the case, the following general causes of prolonged sleep are considered: 1. Narcolepsy. 2. Hysterical somnolence. 3. Lethargy. 4. Catalepsy. 5. Natural and provoked somnambulism. 6. Coma. 7. Sleep due to various causes. In class seven, the author classified those cases which cannot be included in any of the others, and divided this class into two groups: those with no anatomical basis, and those with one. In the latter division the case described in the article is placed. Some consideration is given to the absence of compression symptoms in the third and fourth nerves and peduncles. The author finds the explanation in what he terms "the logic of multiple compression by a basal tumor." The effectiveness or the innocuousness of the compression depends upon two equally important factors: the push (poussée) of the tumor

and the resistance of the nerve. If the push of the tumor is greater, effective pressure follows; if the contrary is the case, the compression is ineffectual because it is inefficient. In this case, owing to the softness of the tumor, due to its semi-liquid state, although developing around the nerve, it was compressed by the nerve instead of compressing it. It is evident that the consistency of the tumor was only one element in this case, as, under more favorable circumstances, mechanically speaking, compression would have followed. The article ends with this general conclusion: the absence of evidence of compression in a structure, if a tumor is suspected from topographical evidence to be present, is not sufficient reason to exclude this diagnosis, which other signs impose upon us. SCHWAB.

- 151 EIN BEITRAG ZUR KENNTNISS DES MENINGOTYPHUS (A Contribution to the Knowledge of Meningo-typhus). A. Hofmann (Deutsche med. Wochenschrift, No. 28, July 12, 1900, p. 448).

Hofmann reports a case of typhoid fever with convulsions in a man twenty-four years old. Clonic contractions first appeared on the right side, in the face and extremities, and later on the left side, and persisted for six hours until death in deep coma terminated them. Other symptoms indicative of meningitis had been present; these are not rare in typhoid fever, but the severe convulsions in an adult are exceptional. Moderate edema and slight clouding of the pia-arachnoid, moderate increase in the amount of cerebro-spinal fluid in the ventricles, round cell infiltration of the pia-arachnoid and of the pial sheaths of the vessels of the brain substance, and a few typhoid bacilli in the subarachnoid space and pia, were found. Hofmann thinks that his case, with the exception of one reported by Tictine, is the only one in which the early lesions of meningitis were found in association with the typhoid bacillus in the meninges. In the cases in which the typhoid bacillus was found in the meninges the meningitis was purulent, and Hofmann's case also it would probably have become purulent if death had not occurred too soon. Hofmann attributes the nervous symptoms to the toxin produced by the typhoid bacillus. SPILLER.

- 152 OBSERVATION DE CHORÉE CHRONIQUE HÉRÉDITAIRE D'HUNTINGDON, EXAMEN HISTOLOGIQUE (Observation on Huntington's Chorea, with Histological Examination). P. Keraud and G. Raviart (Archiv. de Neurolog. 1 Vol., ix, 54).

A case of Huntington's chorea is reported, with autopsy and a microscopical examination by the Nissl method. The results obtained show alterations of the pyramidal cells and the smaller ganglion cells of the cortex. These alterations consist in a perinuclear protoplasmic rarefaction going on to complete degeneration of the cell body. There is also an interstitial and pericellular infiltration of small round cells with small nuclei and an almost imperceptible amount of protoplasm. These changes were found throughout the cortex, but most marked in the motor convolutions of the right side. The alterations in the cells and the infiltration of the small round cells, which they considered neuroglia cells, are likewise found in the spinal cord and especially in the columns of Clarke. MCCARTHY.

- 135 UEBER EINEN FALL VON COMMOTIO CEREBRI MIT BEMERKENSWERTHEN VERÄNDERUNGEN IM GEHIRN (A Case of Cerebral Commotion with Remarkable Alterations in the Brain). G. Hauser (Deutsch. Archiv. f. klin. Med., Vol. 65, 5th and 6th heft., p. 433).

The patient, a man of 56, was found lying unconscious at the foot of a flight of stairs and bleeding from the nose. There was swelling in the right parietal region. The following day he recovered partial consciousness, but he did not react very well to painful stimuli; there was flaccid paralysis of the left hand, and paraplegia, although the patellar reflex remained present. An exploratory incision was made in the right parietal region, which revealed a fracture involving the parietal and temporal bones, and trephining allowed a considerable quantity of fluid blood to escape. Then unconsciousness returned, and finally with rise of temperature the patient died. At the autopsy a fracture was found extending from the base to the convexity of the brain. There was moderate congestion of the cerebral substance of the right hemisphere. The left corpus striatum and optic thalamus were moister and perhaps slightly more swollen than those of the right side; otherwise, the tissues of the brain were normal. Microscopically, there was considerable degeneration of the fibers in the region of the contre coup, and a considerable number of degenerated fibers in the right corpus striatum. The ganglion cells were not particularly affected. It appears from the results of the autopsy that the force of the blow was received by the mastoid angle of the left parietal bone and caused direct injury to the posterior and external parts of the right frontal lobes. There was considerable extravasation in the region of the contre coup, but the most remarkable feature of the whole case was the changes in the central ganglia. This Hauser, after some discussion, concludes must have been the result of cerebral commotion, because there was no crushing of the brain substance, no hemorrhage into the brain substance, even in the part exposed to injury, and therefore the changes can only be ascribed to the continuation of the lesions in a direct line of the fracture.

SAILER.

- 154 UEBER MORPHOLOGISCHE VERÄNDERUNGEN DER VORDERHORNZELLEN DES RÜCKENMARKS WÄHREND DER THÄTIGKEIT (The Morphological Changes in the Cells of the Anterior Horn of the Cord During Their Activity). Joseph Luxenburg (Neurologisches Centralblatt, No. 14, 1899, p. 629).

The experiments and results of earlier researches have stimulated Luxenburg to publish his own investigations into this most interesting question. After narcosis was established in the animal experimented upon, the vertebral canal was opened and the cord cut at the dorsal level, while in the lumbar region a sagittal section was cut. Thereupon the anterior crural nerve of one side was stimulated for one hour with faradism. The sections of the cord were stained with methylene blue and thionin. The results follow: The chief potential energy of the motor cell of the cord is centered in the chromatin bodies of the cell. The activity of the motor cells is evidenced by the degeneration of the chromatin bodies and enlargement of the nucleolus (kornkörperchen?). The position of the nucleus remains unchanged. The products of the protoplasmic metabolism of the cells are released during the activity of the cells. The exhaustion of the cell is accompanied with extensive changes of the chromatic and achromatic substances of the cell.

JELLIFFE.







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